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THE INCREASING IMPORTANCE OF TUBERCULOSIS AS A CAUSE OF OCULAR DISEASE. THE NEWER METHODS OF DIAGNOSIS AND THE TREATMENT.[1]

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THIS paper does not pretend to be a full discussion either of the literature or of the clinical and pathological aspects of ocular tuberculosis, but merely a report of observations which I have been making during the past ten months on the new tuberculin tests, and over a considerably longer period of time on the diagnosis and treatment of tuberculosis of the eye.

Up to a comparatively recent period tuberculosis of the eye has been considered a rare disease. Horner's statistics show one case in 4000; Groenouw's one in 4600; Hirschberg's one in 5000; Eyre's one in 3000; Stephenson's one case of conjunctival tuberculosis among every 1500 patients. More recently Helbron reports that out of 15,000 cases observed in the Berlin clinics, one in every 200 was tubercular, and states his belief that the disease will be observed even more frequently in the future.

[1] Read by invitation at the meeting of the American Ophthalmological Society, July 16, 1908.

Wilder in his address at the recent meeting of the American Medical Association lays special stress on the need of more careful consideration of tuberculosis as a cause of ocular disease.

In 59 cases of chronic uveitis tested subcutaneously with tuberculin by Stock, 61% gave a positive reaction, while in 23 cases of scrofulous disease, 87% reacted.

Hess reports over 50% of positive reactions to tuberculin in 100 cases of ocular disease.

Verhoeff has called attention to the frequency with which cases of scleritis and episcleritis react to this test, and among many others von Hippel and Bull have furthered our knowledge of the frequency of tuberculosis of the eye and of its treatment.

No mention of this subject can be made without referring to Stock's brilliant and exhaustive experimental investigations on animals. By injecting tubercle bacilli into the blood he was able to produce in rabbits, uveitis, scleritis, sclero-keratitis, and finally small nodules in the conjunctiva and chalazion-like swellings in the lids.

It is easy to explain the great discrepancy between the earlier and the later figures on the frequency of ocular tuberculosis. In former years the diagnosis could be made only in one of two ways; either by the finding of tubercle bacilli in the diseased tissue, or by animal inoculation, and it is obvious to what a small number of cases one or the other of these methods can be applied.

The introduction of tuberculin has proved of the first importance but unfortunately its general adoption has been slow, chiefly on account of the accidents which occurred before its dangers were appreciated. Then too the subcutaneous test has its limitations as it cannot well be applied among ambulant patients and thus its use is confined to a comparatively small number of cases. Nevertheless it has added vastly to our knowledge of ocular tuberculosis. The introduction of the new methods of performing the tuberculin test has greatly

extended the applicability of this very valuable diagnostic agent. As to the value of these tests (I refer only to the so-called conjunctival or ophthalmo reaction of Wolff-Eisner and Calmette and to the cutaneous reaction of von Pirquet) it is not necessary for me to speak. For a very complete consideration of the subject I may refer to Wolff-Eisner's work published in the *Beiträge zur Tuberculose*, Band ix., Heft i., a monograph of nearly 200 pages, and 245 references. It must be admitted, however, that as yet there are many unsolved problems connected with these reactions, among which may be mentioned their relative delicacy, their relationship to each other and to the subcutaneous test and the meaning of the various types of reactions which occur.

My observations with them were commenced last September at which time comparatively few articles had been published, and nothing had appeared from the standpoint of the ophthalmologist. My intention therefore was first to determine for myself the applicability of the conjunctival test in the out-patient clinic, and later to investigate from the clinical standpoint the part played by tuberculosis in the etiology of ocular disease. This test was therefore performed on a variety of cases, in many of which there was not the slightest suspicion of a tubercular origin. After sufficient experience had been obtained I limited my observations to those cases in which there seemed to be a likelihood of tuberculosis, and finding that the conjunctival reaction could not be made use of in many instances on account of binocular disease, the cutaneous test was added and where possible in the later cases of my series both tests were applied simultaneously.

For the conjunctival test I first employed a tuberculin prepared by Dr. Baldwin of Saranac, and obtained through the kindness of Dr. John B. Hawes (2d), and Dr. Cleaveland Floyd. My results were not as consistent with this as with the tuberculin supplied by the H. M. Alexander Com-

pany of Marietta, Penna., which I now use. This latter preparation comes sealed in sterile tubes in strengths of ½% and 1%, and I have used the 1% solution almost exclusively. For the cutaneous test I used first Alexander's old tuberculin in full strength and later a 25% solution, as recommended by Wolff-Eisner. For the control vaccination I have employed a solution of glycerine and carbolic acid made up to correspond to the tuberculin. The vaccination has been performed on the left upper arm, the two areas being about three inches apart.

My observations have been made chiefly among the out patients of the Carney Hospital and of the Massachusetts Eye and Ear Infirmary, and I am greatly indebted to the staff of the last-named institution, especially to Drs. F. M. Spalding and L. W. Mansur, for allowing me to make use of their material.

Since the tests were carried out on dispensary patients, it was impossible to make as frequent observations as was desirable, and it is probable that a number of tests have been recorded as negative, which, had the patient been seen in less than twenty-four hours and followed for several days a positive reaction might have been found. This only serves to strengthen the results obtained. Where circumstances permitted, an endeavor was made to have a general physical examination made.

I have tested about 150 cases; 103 of these are reported in this paper. Of these 83 reacted positively, 18 were negative, and 2 were doubtful. The conjunctival test was performed 61 times: 45 positive, 14 negative, and 2 doubtful. The cutaneous test was performed 58 times: 52 positive, 4 negative, 2 doubtful. On 16 patients both tests were performed. On 14, both were positive; once the skin test was positive and the conjunctival negative, and once both were doubtful. Taking up more especially the cases which were thought to be tubercular either on account of the clinical type of the disease, the history, or the general appearance of the patient, I have

93 cases divided as follows: 43 cases classed as phlyctenular disease, because no better general term is available, of which 38 reacted and 5 did not, about 88%; 16 cases of relapsing sclero-keratitis, all positive, 100%; 16 cases of scleritis, 13 positive, 2 negative 1 doubtful, 81%. Nine cases I have classed as uveitis, in most of which the disease was chiefly in the anterior part of the globe. These were selected as possibly tubercular from their general appearance and from the lack of any other apparent cause. All reacted positively. Of 3 cases of interstitial keratitis, 2 were positive, and the third a healed case had 18 months previously reacted to tuberculin subcutaneously, and had suspicious signs in her lungs. In 4 cases, showing vitreous opacities and cloudiness in the lens, one of which showed a peripheral choroiditis, 1 was positive, 2 negative, 1 doubtful. A case of tuberculosis of the conjunctiva gave a positive reaction. The other cases are scarcely worth mentioning as most of them were among my early observations when I was merely experimenting with the reactions. Three chronic dacryocystitis, all negative; two optic atrophy, negative; one typical periodic episcleritis, negative; one corneal ulcer, negative; one in a man with enlarged glands, and in poor general condition, positive; one case of chorioretinitis, positive; an apparently healthy woman with a chalazion gave a delayed positive reaction.

In regard to the reactions themselves there are a number of points which may be of interest. In four cases there was a delayed reaction which did not take place until after 48 hours. All of these patients appeared to be in excellent general condition, which accords with Wolff-Eisner's belief that this is the reaction of the clinically healthy who have an encapsulated focus. Two of these reactions were conjunctival; one was cutaneous, and one showed the interesting feature of a delayed reaction to both tests. In this series of cases, the conjunctival reaction has not been especially characterized by the

redness and swelling of the caruncle which is alluded to by so many writers. Not infrequently a marked injection in the lower bulbar conjunctiva, forming a pyramidal area with its apex at the lower limbus, occurred.

Several of the conjunctival reactions were severe. In one case where the test was made on a supposedly healthy eye, the patient developed a mild episcleritis of three weeks' duration. A more careful examination showed the presence of a previous involvement in the form of a faint corneal nebula. One case of phlyctenular conjunctivitis of the right eye developed a similar condition and also a small corneal infiltration in the previously healthy left eye. This cleared up without incident. Several other phlyctenular cases developed an obstinate conjunctivitis of several weeks' duration. One exceptionally obstinate phlyctenular keratitis of six months' standing developed a severe inflammation in his healthy eye following the instillation. There was very marked scleral injection and a severe infiltration of the cornea. This is only now subsiding after three months. A negro with a tubercular involvement of the conjunctiva of the left eye (bacilli were found in a piece excised) gave a moderate positive reaction in the healthy right eye. He was referred to a tuberculosis clinic for general examination, and there, through inadvertence, received another test drop in the previously tested eye; an exceedingly acute conjunctivitis, with immense and hard swelling of the lids resulted. The eye presented the picture of acute gonorrheal disease. Luckily in ten days' time it subsided without damage. In four cases tuberculin was instilled into a diseased eye. The first has been mentioned, and resulted in a mild episcleritis. A drop of ½% tuberculin was instilled into the diseased eye of the previously mentioned negro, who showed tuberculosis of the conjunctiva. Nothing more than a mild reaction took place. A case which showed vitreous

opacities and beginning cloudiness of the lens in both eyes reacted, but showed no lighting up of the ocular disease. The same was true of a case of chorio-retinitis.

Since there has been considerable discussion as to the value of the reaction obtained after a second instillation of tuberculin, the first test having been negative, I have excluded such cases from my series. In the few cases of this kind which I have seen, the second reaction has always been more marked than the first. Wolff-Eisner, however, believes that the conjunctiva only becomes sensitized to tuberculin in tuberculous individuals, be the disease either open or latent. Likewise I have avoided including any cases which have had tuberculin injections, in order to avoid that possible source of error.

In my hands the cutaneous reaction has apparently been more sensitive than the conjunctival. The figures are misleading, however, as only during the latter part of the work have I used the second test, and therefore chiefly on those cases where a tuberculous infection seemed probable. Then again there was a considerably greater proportion of negative reactions with the first preparation of tuberculin which I used than with the later one.

There have been no untoward results from the cutaneous test in my hands. The reaction has varied in intensity from a very slight redness and swelling around the vaccinated area to a very marked redness and puffiness of some extent, and several times to the formation of a bleb. In no case has the patient complained of pain or difficulty in using the arm, and in most instances he has not been aware that anything has taken place. Wolff-Eisner calls attention to the special value of this test to the ophthalmologist and regrets that it has not come into as much favor as has the conjunctival test. On the other hand various writers have objected to it as uncertain except in infancy, and some regard it as too delicate a test. Wolff-Eisner believes that a positive

conjunctival reaction points to active tuberculosis, while the cutaneous test may appear when there is a healed lesion. The exact relationship between the two has not been worked out as yet, and will not be until many cases have been tested simultaneously by both methods, and the results compared. As Wolff-Eisner points out, it is very desirable that this should be done, and that the subcutaneous test be also used, so that it may be determined in what degree the older and severer method may be supplanted by the newer and more convenient ones. The present weight of evidence tends to prove that the cutaneous test is specific, and of great value. If this be true, it will almost entirely supplant the conjunctival test in ophthalmology, for already in a number of cases, severe disease of the eye has been induced by the latter, while all that can be laid against the former method as yet is the exceedingly rare occurrence of a mild type of lymphangitis, very possibly due to faulty asepsis.

My limited experience has led me to fear the conjunctival test in all diseased eyes, even though the process is healed, and in the healthy eye when there is active disease of the cornea in the other. Also as has been pointed out by several writers, the subcutaneous injection of tuberculin for therapeutic purposes is very likely to light up a fresh reaction in an eye previously tested, and may keep the eye inflamed over a long period of time. The cutaneous test takes but little more time in its application, is at least equally sensitive, and is harmless to the patient.

Since pathological statistics show that a very large percentage of subjects coming to autopsy have active or healed tubercular lesions, it is evident that we should not make a diagnosis of tubercular disease of the eye based alone on a positive tuberculin test. With a view to securing other confirmatory evidence, it has been my intention to have a thorough physical examination made

of each case. This was impossible as my observations were carried out on patients who could not be closely controlled, and most of whom had to be referred to other hospitals for the examination. Many did not return and on others it was impossible to obtain reports. The examinations made were carried out to a large extent by men who have had a very considerable experience with tuberculosis. I have records of 46 cases. Thirty-two of these 46 had physical signs outside of the eye which were regarded as suspicious. Four of the remaining cases gave a tuberculous history, either a parent or child of the patient being affected. Ten cases were negative. In other words, 69% of the cases examined showed some general evidence of tuberculosis.

Let us analyze these cases more closely. Of the 32 which were positive, 16 gave evidence of a pulmonary process. Sixteen cases out of 46 examined showed signs which were suspicious of a tuberculosis of the lungs. In 15 cases out of 46 there was evidence of a tubercular adenitis. One case had a tubercular spine, another had a tubercular knee.

Of the 10 negative cases, 1 was the conjunctival tuberculosis already referred to, showing the bacillus in the excised tissue; 4 were in very poor general condition, but showed no definite signs. Two showed a slight daily increase in temperature.

Of the 32 positive cases 17 had a phlyctenular keratitis, 9 a sclero-keratitis, 3 scleritis or episcleritis, 2 interstitial keratitis, 1 a superficial corneal ulcer. Of the 10 cases with negative physical signs, 2 were phlyctenular, 2 sclero-keratitis, 3 uveitis, 1 chorio-retinitis, 1 interstitial keratitis, 1 tuberculosis of the conjunctiva.

To my mind the phlyctenular series is the most interesting and striking of all. We can no longer doubt that scleritis, sclero-keratitis, and uveitis, are often of tubercular origin, but still there exists a doubt as to the cause of phlyctenular disease of the eye.

My figures, such as they are, point strongly to a tubercular origin. Out of 43 cases, 38 reacted to tuberculin, about 88% (which corresponds exactly to Stock's 23 cases tested subcutaneously). In 19 of my cases a physical examination was made and of these 16 gave some evidence of tuberculosis, nearly 89%,—undoubtedly a greater percentage than will be found among a larger number of cases.

It is said that the phlyctenule is not a tubercular manifestation, first because its pathological structure is not typical of tuberculosis; secondly, because tubercle bacilli cannot be found in it; thirdly, because inoculation into animals does not produce tuberculosis. These objections may be met by the tuberculo-toxin theory by which it is postulated that these lesions are caused by toxins and not by living germs. To my mind the theory of which Wolff-Eisner is one of the chief exponents seems more probable. This states that the question is purely one of dosage, that where only a small quantity of tubercular material is deposited then the pathological changes will not be characteristic and typical tubercle formation will not take place. The first change in a tissue produced by tubercular material may be simply the gathering of lymphoid cells. Unless a greater degree of infection takes place, it seems plausible that epithelioid cells, giant cells, and caseation may all be lacking. Thus we may get tuberculosis without tubercle formation. In this connection it is interesting to note that Wolff-Eisner believes that tuberculin depends for its action on fragments of bacilli which contain an endotoxin, which can only be liberated by lysins manufactured and present in the tissues of the tubercular individual. Furthermore that the tubercular individual manufactures tuberculin in the body and when an excess is present the introduction of more tuberculin from outside fails to excite reaction, as for instance in tuberculosis of the third stage. This view is supported by the experiments of Calmette, Breton,

and Petit, who obtained the conjunctival reaction after the introduction of small quantities of tuberculin, subcutaneously, into guinea pigs, but failed to obtain it when a large quantity was injected.

We know that the instillation of tuberculin into the eye has often produced a typical crop of phlyctenules and has even led to a typical phlyctenular keratitis. It is well recognized that often even in typical tubercular tissue it is difficult to find bacilli and that this tissue introduced into animals will not cause the disease unless a sufficient quantity be taken.

Müller in one case out of 20 infected an animal with tuberculosis by inoculating a phlyctenule. As is well recognized, this disease occurs most often in so-called scrofulous children, and it can be no longer doubted that scrofula is tuberculosis.

Since at autopsy tubercular lesions healed or active are found in from 40% to 90% of subjects, it follows that only in a small percentage of cases, most of them of a severe type, is the diagnosis made during life. There are then many mild cases some of which are not seen by the medical man, and others in which the signs and symptoms are not clear enough to render the diagnosis possible. Most of these patients get well, and only in the event of autopsy is the disease manifest. In a large percentage of cases where it has been possible to make a definite diagnosis of tuberculosis, the disease has been in an advanced stage and cure has been uncertain. This is especially true of pulmonary tuberculosis in which the finding of bacilli in the sputum clinches the diagnosis, but means advanced disease. I believe the same conditions exist in regard to ocular tuberculosis. Formerly only the hopeless cases, leading to the destruction of the eye were recognized, while now we are beginning to recognize the milder types which are curable.

If tubercular disease of the eye is as prevalent as my figures tend to show, it will lie in the power of the

ophthalmologist to take a more active part in the fight against tuberculosis than he can at present. We see hundreds of these cases in our clinics each year. My small series shows that many have signs pointing to tuberculosis in their lungs or elsewhere. These are the incipient cases that the tuberculosis expert wishes to reach. Even if the patient shows no general signs of the disease himself, he may be the means of directing attention to the whereabouts of an active focus of tuberculosis in the community. A few months ago I referred a child with interstitial keratitis and a positive skin reaction for general examination. The report came back that although her general condition was poor, there was nothing definite to be found, but that her mother who was with her and not under medical attention had an active phthisis.

Before taking up treatment, allow me to present one or two tentative conclusions. First, I believe that in ophthalmology it is rapidly becoming possible largely to discard the subcutaneous tuberculin test. In these newer reactions, especially in the cutaneous test, we have a far simpler means of diagnosis, and one that is productive of no harm to the patient. I have seen a number of cases in which to say the least recovery was retarded by the severe local reaction which followed the injection of tuberculin for diagnosis. It seems to me that the use of tuberculin in this way to confirm the diagnosis by local reaction should be employed only with the greatest caution, if at all.

The belief that the conjunctival reaction will be largely supplanted in ophthalmology by the cutaneous test or some other procedure has previously been stated in this paper. Furthermore, undoubtedly, the need for these tests will decrease as we become more and more conversant with the appearance of ocular tuberculosis, for after all a positive reaction alone is but one link in the chain of diagnosis, possibly one of the weakest, and in any case not more

important than the clinical type of a disease history, the general examination of the patient, the lack of another probable cause.

TREATMENT.

I shall endeavor to be as brief as possible and shall say nothing about local measures, as I have already taken up too much of your time. It seems obvious that these patients should be handled like other cases of tuberculosis. The strictness of the treatment should in a general way be proportionate to the severity of the disease. A mild case in a state of fair nutrition may need but little attention, whereas the patient with severe recurrent sclero-keratitis and with signs in the lungs may need a very careful regimen. It is very easy to give advice to a dispensary patient in regard to general hygiene, but it is exceedingly difficult to compel him to do anything for himself unless we have a means of controlling him. We cannot take all of these patients into our hospitals, and most of our eye hospitals, I believe, have poor facilities for handling this disease. I believe the most practical way of treating these cases is by means of the tuberculosis class, and I have been fortunate in being able to refer many of my patients to the tuberculosis class of the Massachusetts General Hospital, which is run in connection with the invaluable Social Service department of that institution, and is under the charge of Dr. John B. Hawes (2d), and Dr. Cleaveland Floyd, to whom I owe a large debt of gratitude. There the patient is carefully examined, weighed, and the temperature taken. He is given explicit directions as to general living, fresh air, amount of food, exercise, and the like. He is given a book in which to make a careful record of his daily life. He is visited in his home by one of the class workers, and is shown how to live in the fresh air, and occasionally he is helped financially when that is necessary. He reports

to me as often as desirable, and once a week at the class.

We have been able to handle our patients very satisfactorily by this method. Of course it will not do for all, as some need still stricter measures before improvement will be noted. Some of these we can refer to the day camp in the warmer seasons of the year, and occasionally a case is sent to the sanatorium.

It seems to me that our first indication is to build up the general health of these patients by every means in our power, and to impress them with the fact that tuberculosis is a chronic process and that a cure is at least a question of months.

I have no statistics as to treatment to present, for of the 40 odd cases which I have followed, none has been under treatment longer than two years, and a number for only a few months. I have under treatment now some 30 patients, among which are 9 cases of sclerokeratitis, 10 phlyctenular keratitis, 5 scleritis and episcleritis, 1 interstitial keratitis, 5 uveitis, 1 tuberculosis of the conjunctiva.

There is no doubt as to the great improvement in the general condition of all these patients, and it has been the greatest satisfaction to observe with what faithfulness they have followed our directions and have continued under treatment for month after month. In most of our cases tuberculin has been used. At first we injected it at the eye clinic, beginning with $\frac{1}{500}$ of a milligram of the old preparation. It did not take long to convince us of the power of this agent. We obtained general and local reactions with as small quantities as $\frac{1}{200}$ and $\frac{1}{100}$ of a milligram, and on several occasions the patient undoubtedly suffered. Since then I have believed that tuberculin should only be administered by the expert. Our patients are now injected once a week at the tuberculosis class, the initial dose being $\frac{1}{100,000}$ of a milligram of new tuberculin. The dose is very gradually and carefully increased and when the patient tolerates

it, it may reach several milligrams. It has seemed to Drs. Hawes and Floyd and myself that these eye patients are especially susceptible to tuberculin, that they are very quick to react locally and get a rise of temperature. In several cases it has been impossible to increase the dose to any great extent. With one, even after months we could not get above $\frac{1}{2000}$ of a milligram. As a rule the production of a local reaction in the eye has seemed to do harm.

My observations on the use of tuberculin in some 30 cases have not convinced me of its therapeutic value. Neither has it seemed to do harm when the proper precautions have been observed. We are still using it and expect to continue. On the other hand, it has been evident that careful and scientific building up of the patient does good and under it and appropriate local measures these patients get well, unless the disease has progressed too far before treatment is instituted.

Allow me to report very briefly a case which shows markedly the results of careful treatment.

L. D., 38 years old, came to the Carney Hospital January, 1907. For years she had been subject to recurrent attacks of ocular inflammation which have materially reduced her vision. The patient had a typical sclero-keratitis with extensive corneal scars, thinning of the sclera, a few posterior synechiæ, V. o. u. = $\frac{10}{200}$. She was taken into the hospital and reacted generally and locally to subcutaneous injection of tuberculin. We gave her tuberculin injections in the hospital and had much trouble in avoiding a reaction. As she did not improve she was referred to the tuberculosis class on March 7th. At this time her weight was 138 pounds; temperature 99.2, and there were suspicious signs at the right apex. Both eyes were inflamed. She was made to reduce her home work to three hours a day, to stay out of doors six hours on a balcony, and she slept with three windows open. She took from three to four quarts of milk in the 24 hours. There was but little improvement in her eyes for two months. Then they quieted down and for a year past they have given

no trouble. December 12, 1907, her temperature was normal; lungs negative; weight increased from 138 to 166 pounds. She was then taking $\frac{7}{1000}$ of a milligram of tuberculin and was the picture of health. Treatment was then stopped except for general precautions and she has remained well since.

As a whole our results have been very encouraging, and I hope to report at length on them at some future time. The only cases which I have had where there has been a permanent diminution of vision are two of uveitis in which there were extensive posterior synechiæ when the patient first came under observation. In one of these cases an iridectomy was followed by an almost complete opacification of the cornea.

THE DIAGNOSTIC AND THERAPEUTIC VALUE OF TUBERCULIN IN TUBERCULOUS EYE DISEASES.[1]

By Dr. ERVIN TÖRÖK, New York.

THE tuberculin invented by Robert Koch in 1889 has made rapid strides in its application for diagnostic and therapeutic purposes. In fact the scientific literature on this subject is almost innumerable. Many refer to it with favor; a great many, however, among them such a foremost scientist as Virchow, criticise it with disfavor. Generally its detrimental effect is attributed to the alleged action that it renders bacilli in a state of repose, mobile, thus spreading them about the entire system; consequently it is not only therapeutically not beneficial, but, by causing generalization of the local ailment, may terminate fatally.

This is mainly the cause that the great expectations which were linked to the remedy at the time of its discovery have vanished within a few years, and that not much reference is found in the medical literature of late. It seems to be plausible that these unfavorable, nay sometimes fatal, effects must be attributed to excessive doses (there are cases where 30–40, even 50*mg* were used) and unsystematic application. This is corroborated by the fact that, lately, where the application of tuberculin was confined to smaller and more

[1] Read in part at the meeting of the Ophthalmological Section, N. Y. Academy of Medicine, May 18, 1908.

carefully applied doses, no such detrimental after-effects were reported. To illustrate this, I shall refer to Dr. Beck's statistics, who applied tuberculin for merely diagnostic reasons and obtained 1525 positive reactions out of 2508 injections, and did not observe a single case with detrimental effect. A like report can be found from other medical authors, so that presently we may say that the general opinion of the tuberculin culminates, that if carefully dosed, and applied in minimal quantities, rising to a certain maximal standard, it not only proves to be safe, but, from a diagnostic as well as from a therapeutic point of view, it is an excellent and in many cases indispensable remedy.

Von Hippel in his treatise published in 1904 was first to recommend again the availability of tuberculin in cases of ocular tuberculosis, in which he especially emphasizes its merits, based upon his ten years' experience with tuberculin, warmly recommending its use to the medical profession.

Tuberculin is applied for diagnostical and therapeutical purposes.

To ascertain whether an eye disease is of tuberculous origin or not, meets often with difficulties, as tuberculosis may attack either part of the eye, and may take its course by manifesting itself through various acute and chronic inflammations.

This is the reason that no deductive diagnosis can be made by the mere clinical symptoms, but generally some other circumstances have to be considered, and we have to resort to secondary means which may be helpful to lead us to a positive diagnosis. To illustrate this I shall quote Nesse's case, where a 33-years old, strong individual, afflicted with lues, suffered from a typical irido-choroiditis luetica, and, after the enucleation was performed, the pathological anatomical examination of the eyeball showed that the choroiditis was of tuberculous origin.

At first great care must be taken as to a minute anamnesis, and, in consideration of the heredity of tuberculosis, special stress must be laid upon the family history. We must well ponder the age of the patient, his general appearance and condition of nutrition, and make a thorough examination of the entire system, whether or not other organs are affected with the disease. To ascertain the diagnosis, we may excise a small part of the diseased tissue and inoculate it in a rabbit or guinea-pig, or we may try to find tuberculosis bacilli either in the excised tissue proper or from the sloughs taken from the surface of the diseased part. A very important factor is the histological examination of the excised tissue, or eventually the Arloing-Courmont sero-diagnosis, and finally the best and most reliable agent is tuberculin. (Here I wish particularly to emphasize that either one of the given tests may fail; thus we cannot accept or eventually reject the theory of the possible presence of tuberculosis by entirely depending upon the positive or negative result of a single test, but we have to base the diagnosis upon the collective results of several of the enumerated tests. And this implies the difficulty and uncertainty of a proper diagnosis.) These are the paramount principles by which I have been led in establishing a diagnosis.

There is no doubt that in these days tuberculin is the most important and most reliable agent from a diagnostic point of view; this was acknowledged long ago by all who availed themselves of it, and it was unanimously hailed by the Tuberculosis Congress in London in 1901 as an excellent, reliable, and safe remedy. Its reliability was also elucidated at the said Congress by E. Franze, stating that he administered 55 tuberculin injections, 45 of which showed positive reaction,—the balance of 10 however did not; 29 of the former 45 patients at post-mortem examination showed evidences of tuberculosis, whereas in 5 of the above mentioned 10 no tuberculous lesions

were found. I deem it important to mention that it is generally used in veterinary practice, where the propriety of the established diagnosis by reaction can always be ascertained by autopsy. The Third International Veterinary Congress unanimously declared tuberculin to be the best diagnostic agent up to date.

Favorable reports on the value of tuberculin for diagnosis in eye diseases have been published by Haab, Pflüger, Morax, and Chailloux, furthermore by von Hippel in his widely known article, and the Congress of Heidelberg in 1905 recommends it too. Based upon experiments at the University of Tübingen, Reuchlin says that if a patient with a suspicious inflammation of the eyes reacts in the typical manner to small quantities of old-tuberculin (1–3–5*mg*) the existence of an active tuberculosis is proven. This agrees with my opinion. All the cases which I have treated and which reacted to tuberculin proved themselves to be cases of tuberculosis and were completely cured after treatment with Tuberculin R. Following the recommendations of the above mentioned authors, I always used for diagnosis Koch's old-tuberculin (T. V.) diluted with 0.25% phenol solution. The method is the following: after the patient's temperature had been taken every two hours for a few days and found to be not over the normal, 1*mg* old-tuberculin was injected subcutaneously in the interscapular region at 8 P. M. The taking of the temperature bi-hourly must be resumed on the following morning. Should the patient not have shown any reaction after the expiration of 48 hours, 3*mg* are injected, and 5*mg* 2 days later if the result has again been negative. Children were submitted to an exactly identical procedure. I have strictly observed the 2 days' intervals, as many patients showed reaction only from 24 to 36 hours after the injection had been made.

The reaction may be local or general. The general reaction is the more important; from a few hours, up to

1 or 2 days, after the injection, chills seize the patient, the temperature rises and then suddenly falls after a few hours.

The subjective symptoms vary. Some patients complain of headache, nausea, dizziness, vomiting, and pain in the extremities; the hands are heavy, and the use of the lower extremities is almost prevented by the sensation of their heavy weight. Others, however, where a positive reaction sets in after administering a comparatively small dose, scarcely complain of any subjective symptoms. In considering a positive reaction, the height of the temperature is not so important as the characteristic curve of temperature which manifests itself in a sudden rise and a sudden fall.

The local reaction is not so frequent and can usually be observed when large doses (5*mg*) are administered. The symptoms vary according to the pathological condition of the eye, and consist in an increase of the inflammatory symptoms. Œdema of the eyelids is noticeable after the injection in cases of tuberculosis of the conjunctiva; the conjunctiva discharges a copious mucopurulent secretion and becomes strongly swollen and succulent; the yellowish nodules increase in number and in size and become more yellow. The local reaction in tuberculous scleritis manifests itself chiefly in the increase of the ciliary injection and pain; the yellow nodule, if present, increases in size and is often surrounded by new and smaller nodules. In tuberculous iritis, with the increase of the ciliary injection we may observe that the iris which previously showed only a few nodules is covered with many new ones, and the opacities of the vitreous become denser, and the visual acuteness diminishes. In tuberculous choroiditis we might observe that the small yellowish tuberculæ of the choroid increase in size and number.

For therapeutic purposes I always employ new-tuberculin (T. R.). This is given in subcutaneous injections

commencing with $\frac{1}{1000}mg$, increasing the dose gradually by $\frac{1}{1000}mg$ each injection, up to $\frac{1}{100}$, then by $\frac{1}{100}mg$ up to $\frac{1}{10}$, and then from $\frac{1}{10}$ up to $\frac{5}{10}$, *i.e.* $1mg$, by $\frac{1}{10}mg$ each injection, according to the method suggested by von Hippel. I have never exceeded $1mg$. As a reaction might show itself on the following day, the injections should not be given more frequently than every other day, or 3 times a week. Children were submitted to the same dosage and procedure. The patients are instructed to take their temperature every 2 hours and keep a temperature record which they must bring to the physician. In my experience this practice is free from risk and more advantageous than the hospital treatment, as the patient has the benefit of fresh air, which is of course an important factor in the treatment of tuberculosis. Should the injection be followed by a reaction, the patient has to remain in bed and wait until it abates. It rarely lasts longer than 6 to 8 hours. The patient has to report for another injection only when the fever has subsided. A rise of temperature was rarely observed in cases where the increase in dose was slow; should it however occur, the dose ought not to be increased, but the original dose given repeatedly until the patient's system endures it without reaction. In case the reaction is intense and the fever high, the dose must be diminished. This reaction is rather uncertain and resembles the typical general reaction observed upon administration of old-tuberculin, and is often accompanied by headache, dizziness, nausea, vomiting, debility, sluggish sensation in the extremities, and sometimes local reaction in the eye.

I have often experienced that, after having administered the same dose several times in succession, the patient reacted with high fever at one time, whereas on other occasions he was absolutely free from fever, despite the fact that these injections were made from the same solution, under the same circumstances, and at the same

hour. The exact cause of this peculiarity I do not know; it might be that, perhaps through injecting the tuberculin incidentally into a vein, its rapid conveyance and distribution into the system might cause this fever. Simultaneously I must declare that I am not afraid of these febrile reactions, as aside from some disagreeable subjective symptoms, lasting for a few hours, I have never experienced any detrimental after-effects; on the contrary, I can report only rapid improvements in many cases. I do not want to say that this fever is desirable, but only that in my experience it is harmless. At the same time these reactions should be avoided. I generally inject alternately in the upper arms. Generally 8 to 10 hours after the injection, the surface of the skin where the injection has been made shows a sharply defined swelling, indurated, red, warm, and painful, causing some discomfort to the patient, most especially when moving the arm. This disappears after 1 or 2 days, the skin becomes normal, and just a small indolent thickening beneath the scar indicates the point of the last injection. Should the swelling be very intense and sensitive, the pain can be abated as well as the absorption accelerated by making an application with a cloth soaked in cold water and covering it with a dry cloth. This may be changed every 3 to 4 hours. Some patients do not show any reaction at all and stand the injections without any local reaction. In other cases this local inflammation increases and an abscess forms, which after incision discharges dense yellowish-white pus. This is not the result of an accidental infection, as I have invariably observed the strictest aseptic precautions and the pus has been found sterile on bacteriological examination. Some patients show a particular tendency to form abscesses. In 2 patients out of 21, abscesses were produced; the tendency to the formation of abscesses, however, ceased after a certain length of time. This seems to me to indicate a certain idiosyncrasy, which

the patients gradually overcome upon persistent injections.

According to my experience, tuberculin applied in doses and in the manner above described was never detrimental to the general condition of the patient; on the contrary, the general appearance of the patient showed great improvement, the weight increased in consequence of better appetite,—one of my patients gained 12 lbs. within 2 months' treatment. In a great many cases of ocular tuberculosis the ailment is solely of local character, videlicet the physical examination cannot ascertain a tuberculous condition of other organs. Notwithstanding, should there be tuberculosis present in the lungs or other organs, said treatment proved but beneficial. One of my patients suffered from pulmonary tuberculosis and showed visible improvement after having been treated for 4 months with Tuberculin R. In another patient's case the osseous tuberculosis was perfectly cured.

The ocular tuberculosis under treatment shows a gradual retrogression. In cases of tuberculosis of the conjunctiva, the œdema of the eyelids gradually disappears, the ulcers of the conjunctiva clear up and cicatrize, the large exuberant granulations shrink, and the small yellowish nodules which are scattered about disappear, leaving no traces, so that at the termination of this treatment, aside from some scars on the conjunctiva, the surface is smooth and thin. The pannus on the cornea which is often associated with this ailment also disappears. The smaller or larger nodules on the sclera disappear, sometimes leaving a small gray spot in their place, but as a rule they are absorbed and vanish, leaving no traces. Opacities of the cornea clear up rapidly, and not only the recent infiltrations but also the older scars which have existed for several months become thinner and more transparent.

There were also cases where the cornea, transformed into a white opaque mass, through which the iris was ab-

solutely invisible, became in consequence of the treatment much more transparent and the patient gained some vision.

One of my patients suffering from a grave interstitial keratitis, whose vision was reduced to the mere perception of light, gained vision of $\frac{20}{30}$. Another case of scleritis and keratitis sclerotisans showed an improvement of the vision from $\frac{20}{200}$ to $\frac{20}{40}$.

In case of tuberculous iritis, the small nodules, surrounded by newly generated blood-vessels, disappear, and the large ones are absorbed, leaving a small grayish spot in their place. Exudations constituting thick pupillary membranes may disappear to a remarkable degree. In one of my cases of tuberculous iritis, the pupil was covered by a thick membrane so that the fundus was absolutely invisible. At the termination of the treatment, the fundus was clearly visible and proportionally great improvement of the vision was observed. Dense opacities of the vitreous, which reduce the vision to a mere qualitative one, disappear entirely. As an illustration, I wish to refer to one of my patients, suffering from tuberculous irido-cyclitis, whose vision improved from mere perception of light to $\frac{20}{15}$. Similar good results can be obtained in tuberculous choroiditis disseminata, where of course the atrophic patches cannot be revived, but the recent yellowish spots clear up very nicely, the vision improves considerably, and the disease is arrested. One of my patients suffered from disseminated choroiditis for the last 6 years and was also myopic, could count fingers only up to 8 feet, and no improvement was obtainable by using concave glasses; aside from haziness of the vitreous both fundi were densely scattered with various-sized yellowish and white patches, and both optic disks showed an incipient atrophy. Since the beginning of the disease it gradually progressed, causing diminished vision. Upon treatment the disease not only ceased, but his vision improved so that by means of a -3.50 sph. lens he sees $\frac{20}{20}$.

The treatment must be kept up until all inflammatory symptoms have subsided. The ulcers of the conjunctiva must have completely cicatrized, the exuberant granulations as well as the yellow nodules completely vanished, and the conjunctiva become smooth and thin; the sclera entirely white with no injection or nodules; the infiltrations and recently generated blood-vessels of the cornea disappeared, the corneal deposits absorbed, the nodules of the iris gone, the opacities of the vitreous and the yellowish patches of the choroid absorbed. This generally requires considerable time, so that the treatment may last 5–6–8 or more months. As a rule, I do not consider the cure terminated even with the disappearance of all inflammatory symptoms, and do not cease with the injections, but administer one bi-weekly injection for about 2 to 3 more months.

Twenty-one ocular tuberculosis cases were treated by me according to this method; among them 9 tuberculous scleritis, 5 tuberculosis of the conjunctiva, 4 iritis, respectively irido-cyclitis tuberculosa, 3 choroiditis tuberculosa, and 1 tuberculous interstitial keratitis.

Barring one case of tuberculosis of the conjunctiva and another of irido-cyclitis tuberculosa I have invariably obtained satisfactory results. The case of tuberculous conjunctivitis referred to existed for 6 years and was very neglected and in a very advanced stage, with extended scars and ulcers of the conjunctiva and thick pannus on both corneæ, and was complicated with lupus of the nose and extended cuticular lupus. The other was a case of tuberculous irido-cyclitis where I faced a conglomerated tubercle of the iris and ciliary body of old date and the size of about a bean, which despite of one month's treatment did not diminish, but, increasing in size, perforated the cornea and sclera so that I deemed it necessary to remove the eyeball. At this point I wish to remark that a chronic disseminated tuberculosis gives invariably a better prognosis than a conglom-

erated one, whose behavior is similar to a new growth.

Regarding the permanency of the cure and the relapses, I wish to say that perfect and permanent cure can be attained by availing ourselves of the above enumerated methods; this conclusion of mine is also confirmed by the fact that among 19 recovered and cured patients, some of whom for 3 years, the majority however for 2 years, not in need as yet of any medical aid, there were no relapses whatsoever.

Briefly summing up the above, I wish to state that, according to my opinion and experience, from a therapeutic and diagnostic standpoint tuberculin is an excellent preparation.

From a diagnostic point of view I consider the old-tuberculin (T. V.) one of the best and most reliable agents, and I may say that my belief is that, when a patient's eye disease suggests tuberculosis from other circumstances (age, clinical picture, heredity, etc.), and a typical general reaction is present after administering small quantities of T. V. (not exceeding 5*mg*), we can positively make the diagnosis of tuberculosis, even when we do not get local reaction in the eye itself.

From a therapeutical standpoint I consider the T. R. an excellent remedy which, with due care and when properly applied, cannot cause any detrimental effect, either to the eye or with regard to the general condition; on the contrary, it may prove helpful, as it did in most of my cases, to lead to perfect recovery and permanent cure. Naturally, like everything else, the acting potency of the T. R. is also limited, and we sometimes may not attain positive results in neglected and protracted cases, still, considering the innocuous propensity of the tuberculin, a trial can be made even in those cases, as this is the only possible means to attain a successful cure.

In addition to the 16 cases published in the ARCHIVES

OF OPHTHALMOLOGY, vol. xxxvi., No. 5, 1907, I now report 5 new cases treated with T. R. in the N. Y. Ophthalmic and Aural Institute.

CASE 1.—Female, 47 years of age, who was suffering from episcleritis in both eyes for the last two years. Her father, after having suffered from some pulmonary ailment for 9 or 10 months, died in his 48th year of age; 3 sisters and 1 brother died from pulmonary ailment. Her husband is sound. Married for 10 years and had no children. She was always in good health up to her present illness. No tuberculosis can be ascertained in any part of her body. A typical general reaction was perceptible after the first administration of 0.001*g* T. V. In consequence of treatment with T. R. there was perfect recovery, inasmuch that no relapse was perceived for more than one year since the treatment. Formerly episcleritic attacks were noticeable every 5 to 6 weeks. Altogether 37 injections were made.

CASE 2.—Male, 22 years old, who was afflicted with a chronic irido-cyclitis of the left eye; negative family history. He never suffered from any serious illness. This ailment began 11 months previous to the treatment; his vision gradually diminished so that at the time of his first calling upon me he could count fingers only from a distance of one foot. No tuberculosis can be ascertained in any part of his body. A typical general reaction was perceptible after the administration of 0.001*g* T. V. Upon treatment with T. R. he recovered totally. The inflammatory symptoms subsided and the dense opacities of the vitreous were absorbed. His vision is at present $\frac{20}{15}$. The treatment lasted from March to September, and the patient is perfectly well since. Altogether 35 injections were given.

CASE 3.—Male, 29 years of age, afflicted with a chronic irido-cyclitis and chorio-retinitis for the last 6 years. Negative family history. He was always sound; no tuberculosis found in any part of his organism. Left pupil closed by a membrane; opacities of the vitreous and yellowish-white patches joined together in the choroid of both eyes. Optic disks pale, sclerosis of the vessels. Vision: R. fingers at 10, L. at 3 feet. After typical general and local reaction he

received T. R. injections for period of 8 months. The vision of the R. eye improved to such extent that he sees $\frac{20}{15}$ with a — 3.50 sph. The pupillary membrane occludes the vision of the left eye, but also here the vision improved from fingers at 3 feet to fingers at 15 feet. I believe that the sight of the left eye could be still further increased by iridectomy. The vision has been stationary for the last 10 months.

CASE 4.—Male, age 24, suffering with a grave interstitial keratitis of 5–6 weeks' duration. Negative family history. There is no suspicion of lues in his history, and he was always in good health up to his present illness. Vision on admission was limited to light perception. Reacted positively to 0.001*g* T. V., after which T. R. injections were given for a period of 8 months; 56 injections in all. All inflammatory symptoms subsided, cornea cleared up, and the vision was restored to $\frac{20}{30}$. Marked improvement was also noted in his general condition.

CASE 5.—Female, age 36. Negative family history; 4 years ago had an inflammation in both eyes which destroyed the sight of the right eye, but vision in left eye returned; 3 weeks ago left eye became inflamed, since which time she has been unable to see. On examination, right eye shows the results of a previous irido-cyclitis with secondary glaucoma. Left eye shows an active irido-cyclitis, with occlusion and seclusion of the pupil and secondary glaucoma. Iridectomy was performed immediately to relieve the tension. 3 weeks later patient reacted both locally and generally to 0.001*g* T. V. The administration of T. R. was then continued for a period of 6 months. Altogether 48 injections were given. Result: a gradual disappearance of all inflammatory symptoms and restoration of a greater part of vision. No relapse. This patient has been under observation nearly a year.

A CLINICAL STUDY OF TUBERCULOSIS OF THE FUNDUS OCULI.

By Dr. W. KRAUSS, Marburg, and Dr. A. BRUECKNER, Wuerzburg.

Abridged Translation from Vol. LVII., No. 3, German Edition, of these Archives, by Dr. Percy Fridenberg, New York.

(*With two colored drawings on appended Plates X. and XI.*)

THE observation, in rapid succession, of two cases of this rare condition by one of us (Krauss), and the absence of representations of anything at all similar in any atlas or text-book, have induced us to report the following cases in full, particularly as they represent some interesting features in the matter of the diagnostic importance of test-injections of tuberculin:

Case 1.—Marburg Clinic. A quarryman, age 19. Since July, 1904, patient claims, vision of right eye has been dim. It was first noticed after a spark from a grindstone had flown into the eye. Admitted, April 25, 1905. Family history: Mother died of some lung trouble. Otherwise negative. Previous history, negative. Physical examination: Patient tall, slender, and anæmic. Suspicious signs of tuberculosis at the apices of both lungs. Examination of blood and urine negative.

Examination of Eyes: O. D., normal. V. =6/5. Emmetropia. Fundus and accommodation, normal.

O. S., lens and cornea, clear. Fine, floating opacities of the vitreous.

Ophthalmoscopic examination (Plate X. Reversed image, after a sketch by Krauss): A large whitish area is at

once noticed at the posterior pole. The disc is not quite clear, grayish-red, but the margin is sharp. Vessels, normal. Close to the disc, and framing its temporal margin with a sickle-shaped prolongation, the white area mentioned appears and extends upwards and to the nasal side. At the macula it surrounds a small area, about the size of the disc, of normal red retina, which is slightly (+1.5 D) prominent. Here there are one or two deposits of pigment in the retina. Between the macula and the nerve-head there is a wing-shaped process about 2 P. D. long. Beyond the macula, about at the level of the lower margin of the disc, the large white area begins and extends far upwards. Its breadth is twice that of the disc. At several points there are crescentic prolongations. This area forms a ring around an oval area of partly reddish and partly gray-green retina which is decidedly prominent (2.5*mm*) and undoubtedly is detached retina. The detached area shows absolutely no trace of tremulousness or floating and gradually descends to the level of the surrounding white area. It presents a large number of retinal hemorrhages, old and recent, generally in connection with small arteries.

The large area looks as if it had grown by coalescence of smaller plaques, a number of which are seen in the fundus. There are no pigment deposits within the white ring.

Vision was reduced to counting fingers at 2–3*m*. The field showed loss of entire upper half, with contraction below. No disturbance of color sense. Tension, somewhat reduced.

As tuberculosis was suspected, the patient, who had been under treatment with subconjunctival salt injections, received an injection of 0.5 *milligram* of old-tuberculin. Temperature, which had been normal, rose to 37.2°C. Two more injections were given, followed by a rise to about 38.2°C., but the general condition of the patient became worse. The iodides and salt injections were now used. There had been no change in the function, but each injection of tuberculin was followed by decided changes in the fundus, increase in the retinal hemorrhages, particularly in the upper portion. The fundus also became more cloudy, and the vitreous more opaque.

The patient was discharged, unimproved, in July, but seen

at intervals of a month. At the last examination, in April, 1906, the white area and spots had progressed so far upward that no normal retina could be seen. Some new hemorrhages were noted, and some of the old exudates had united to form larger patches.

Case 2.—Wuerzburg Clinic. Apprentice, aged 14. Admitted Dec. 11, 1905. Patient has noted dimness of vision of left eye since 3–4 weeks following acid burn. There is some history of lung trouble in the family, but the patient has no cough or night-sweats. Physical examination, negative. O. D., normal. V. =6/5. Emmetropic. Field normal. O. S., externally normal. Lens clear. Floating opacities in vitreous. Ophthalmoscopic examination: Disc (Plate XI) somewhat congested, not sharply defined. Vessels, a bit distended. A large white area, beginning at the disc, extends upwards and inwards. The color is yellow-white, with some grayish regions like shadows. This white patch reaches to the mid line of the eye and gradually merges into the normal retina by breaking up into yellow white dots which separate more and more. The temporal half of the retina appears normal. The white area is about 2.5 D. more prominent than the disc, but toward the periphery there is a detachment nearly *3mm* deep. On its summit there are a number of hemorrhages, mainly from small arterial twigs.

Vision in this eye was reduced to counting fingers at *1m*. T. N. Clinical examination showed some anæmia, but no evidence of tuberculosis.

The patient was put on iodides, and two injections of old-tuberculin, *2mg*, were made without any general reaction, but with some changes in the fundus picture. The hemorrhage had increased, and a layer of exudation noted on the summit of the detached area had become more prominent. The injections were discontinued, and subconjunctival injections of saline solution, sweats, aspirin, and hot compresses used. No change was noted.

On Feb. 2, 1906, an increase of the layer of exudate was noted and there were several fresh hemorrhages in connection with arterial twigs. The picture is otherwise unchanged. Tension, slightly lowered. Discharged, unimproved. About four weeks later, examination showed a marked increase of

vitreous opacities. Fundus appearance and vision about as before.

Readmitted, Feb. 27, 1906. Marked opacity of vitreous. White area somewhat more prominent than before. Detached area as before. Pre-retinal exudate has become more extensive, covering all the hemorrhages on the summit, except some recent ones. The entire process has advanced above toward the temporal half of the retina. The small yellow deposits in the lower nasal half have become still more numerous. Transillumination, positive. Tension, slightly reduced.

These two cases show a marked similarity in the picture and course of the disease, and there is hardly a doubt that they depend on the same pathological cause. Both patients were young men with a hereditary taint of tuberculosis. Both date the ocular disease from an injury which was actually of no importance, except, perhaps, in attracting attention to the condition of the eye. The involvement of the left eye only, the marked disturbance of function, painless course, loss of central vision, with corresponding defect in the field of vision, and the limitation of the disease process to the posterior half of the globe, were striking features in both cases, and there was similar involvement of the vitreous in each.

The details of the fundus changes, too, are quite alike. The condition of the disc and vessels, showing that there was no primary disease of nerve or retina, the presence of multiple hemorrhages, the yellow-gray deposits of varying size, the small, pin-head-size protruding white dots, the retinal detachment, with a surrounding zone of white discoloration, the lack of pigment deposits, are the same in both.

Finally we have the appearance of fresh hemorrhages after the injection of tuberculin, as the expression of a local reaction. There are few dissimilarities. In the first case there was extraocular tuberculosis, in the form of consolidation of both pulmonary apices, while nothing

of the sort could be found in Case 2, which also failed to react to tuberculin.

Some minor differences in the ophthalmoscopic picture could easily be explained by individual variation in the pigmentation of the fundus, the relative size and arrangement of the vessels, and the stage or age of the disease process, which was probably of longer standing in Case 1. The condition described is certainly a rare one, which we believe to be due to tuberculosis, of chronic course, affecting the retina mainly, and the choroid to some extent.

Siegrist (1) reports a case much like ours which had been previously described, under a different title, by Weltert (2) and Schiess (3). The fundus presented the same large brilliant white mass surrounded by grayish-white dots and round deposits, and a bluish-gray portion with hemorrhages. The disc and vessels were affected; the veins, particularly, being markedly engorged and tortuous. The patient had uncompensated mitral insufficiency. Siegrist thinks the vascular changes were due to thrombosis, and the white areas were the expression of retinal œdema, while the changes observed gave no evidence of an inflammatory process. Mellinger (4), in discussion, stated that when he examined the patient she showed fundus changes of retinitis circinata only, and no cardiac lesion could be made out. This might allow us to conclude that the vascular changes in the fundus were secondary, developing in the course of a chronic inflammatory process involving the retina, and perhaps the choroid as well. This seems to have been the interpretation of Schiess. In this case, too, there was tubercular inheritance.

The case reported by Carpenter and Stephenson (12, Fig. 25) resembles ours except that the choroidal changes were most prominent. The authors believe the retinal detachment to have been caused by a conglomerate tubercle of the choroid, and that the small white dots, re-

sembling those seen in albuminuric retinitis, represented miliary tubercles of the deeper retinal layers.

As to the detachment in our case, this might as well have been due to choroidal neoplasm. Sarcoma usually presents a different picture, but Schieck has noted that the typical appearance may be obscured by a choroiditis with plastic exudation. The negative result of transillumination would exclude this condition in our case.

Our study of the literature shows that cases have been published as tuberculosis of the retina on the strength of the clinical picture alone, while others of probable retinal tubercle appeared under other titles. In our case the diagnosis of tuberculosis was based on the heredity, the course of the disease, and the reaction to tuberculin. The localization in the retina was shown by the local tuberculin reaction in the form of fresh retinal hemorrhages and by the clinical course of the disease.

Clinical studies on the therapeutic and diagnostic significance of test-injections of tuberculin have convinced us that tuberculous disease of the eye, particularly of the fundus, is not at all uncommon. Such a condition can only be demonstrated by the occurrence of local reaction in the fundus in addition to, or in place of, the general reaction, whereas the latter alone has no significance as regards the presence of a possible intraocular tuberculosis. The development of fresh hemorrhages, the intimate relation of this process to blood-vessels, and the deposits of inflammatory exudate in their immediate neighborhood, show the same hematogenous origin as the well-known experimental variety. It is possible that the retinal changes were secondary to choroidal disease.

In regard to the ophthalmoscopic picture we have already noted the probability of the slight vascular changes in the retina being of a secondary character keeping pace with the extension of the inflammatory process.

The main characteristic of the ophthalmoscopic picture is a large yellowish-white mass spreading out in all directions from a detached area of retina. This may cover a tuberculous tumor situated in the choroid and possibly covered by a subretinal exudate. The discolored yellowish-white part of the retina may have originated in an exudate or in a cicatricial or fibrinous degeneration of the posterior layers of the retina. The latter could easily be sufficiently thickened in this way to cause an ophthalmoscopically visible difference of level. The numerous irregular yellowish-red deposits and the larger patches are situated deep in the retina, or at least below the level of the nerve-fibre layer, as vessels can be continuously traced over them. The grayish-white, sharply defined, round dots are farther forward and can be seen to raise up the surface of the retina and small vascular twigs crossing them. It is a question whether the first variety represents accumulations of round cells, coagulations of fibrin, fatty degeneration of the nuclear layers, or degenerative changes in small nummular hemorrhages.

The small prominent deposits may have a vascular origin and we are by no means sure that they are not miliary tubercles. The almost complete absence of pigment changes would exclude any active involvement of the choroid, although this may be due to the obscuration of choroidal changes by the opacity of the retina so frequently noted in the early stages of a process involving both retina and choroid.

Case 2 was seen again on March 4, 1907. The fundus picture had changed but little. The small white prominent spots had disappeared entirely, having possibly coalesced to form large patches, several of which were seen. The detached area appeared firm and somewhat nodular. This conglomerate character of the exudate in, or behind, the retina speaks strongly in favor of the diagnosis of tubercle.

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PARALYSIS OF THE ABDUCENT NERVE ASSOCIATED WITH OTITIS MEDIA.

By Dr. ARNOLD KNAPP, New York.

THE unusual association of abducent paralysis with an apparently uncomplicated inflammation of the middle ear has in recent years been observed in a number of cases. In the following case this association was particularly clear, and the other symptoms are of interest as throwing some light on the probable connection.

Previous History.—E. B., 5 years old, a well developed and previously healthy child, after an attack of grippe, suffered from headache and pain in the right ear. This persisted for two weeks and was associated with unusually severe pain in the right half of the face and in the right eye. The gums on the right side of the mouth were so sore that she could not chew. The mother then noticed that the right eye turned in.

On examination February 29, 1908, the right eye is in a convergent position. No outward motility beyond the middle line. Homonymous diplopia. Vision normal. Sensibility unchanged. No history or signs of herpes. Field normal. Under atropin, H+0.5. Pupils and eye-grounds normal. The right ear-drum is red, verging to white; slightly bulging; no perforation. As there is no pain at present and the ear condition is on the way to recovery, no paracentesis is done. The child is kept quiet; hot-water bag and calomel in small doses are prescribed.

March 17, 1908, general condition much better. The ear-drum is nearly normal. The ocular paralysis is unchanged.

March 25th.—The right eye can now be turned outward 20°.

April 8th.—Motility not quite to external canthus; homonymous diplopia.

May 14th.—Motility normal. No diplopia. An intercurrent attack of headache and earache but no return of the ocular paralysis.

June 11th.—Well.

The symptoms in this case, beyond those of an acute otitis media of moderate severity, were paralysis of the right external rectus and pain in the right eye, cheek, and gums, and inability to chew. The latter group are suggestive of a lesion of the Gasserian ganglion. The involvement of the ganglion was transitory; the paralysis of the sixth nerve was recovered from in two and one-half months. The proximity of the Gasserian ganglion to the sixth nerve at the apex of the petrous pyramid points to that as being the seat of the anatomical process. It is well known that this region can be involved by an extension of a purulent process from the tympanum along the cancellous bony tissue surrounding the osseous labyrinth directly from the inner tympanic wall, or through the carotid venous plexus, that network of veins which surround the carotid artery on its way from the anterior tympanic wall to the cavernous sinus.

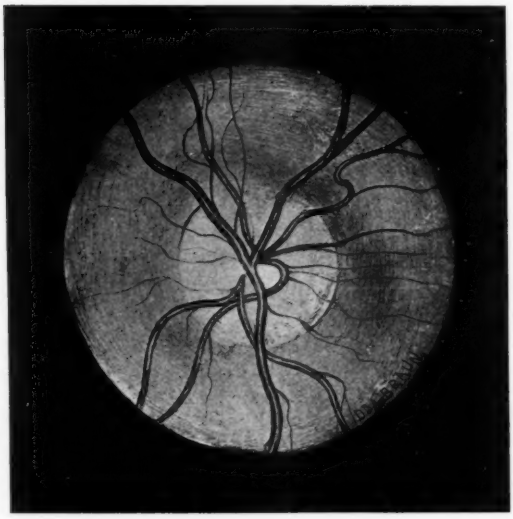
Gradenigo (Intern. Otological Congress, Bordeaux, 1904) was probably the first to draw attention to the connection of abducent paralysis and otitis, and in otological literature the paralysis of the external rectus, otitis, and headache constitute Gradenigo's syndrome.

Many theories have been advanced to explain this association in the cases of otitis uncomplicated with meningitis or sinus thrombosis. The ocular paralysis was generally regarded as reflex until Terson (*Annales des maladies de l'oreille*, July, 1906) offered a more plausible explanation by suggesting the above-described extension along the carotid canal. The most recent paper on the

subject is by Baldenweck (*Ann. d'oculistique*, April, 1908). This author, after an exhaustive review of the recorded clinical cases and autopsy findings regards the anatomic lesion as an ostitis at the petrous apex, the extension taking place along the cancellous tissue from the tympanum to the apex of the petrous pyramid. He is led to this conclusion from the autopsy findings in fatal cases where an involvement of the Gasserian ganglion resulted from caries at the petrous apex. The extension along the carotid venous plexus has never been demonstrated, and according to this author, though it may explain a lesion of the sixth nerve, the associated involvement of the Gasserian ganglion is not so clear.

It is interesting to observe that this complication of otitis has generally occurred in cases with insufficient drainage; in our case perforation had not taken place. The prognosis varies and is good where no other symptoms of intracranial complication are present.

ILLUSTRATING DR. TYSON'S CASE OF
CYANOSIS RETINÆ.



LEFT FUNDUS, UPRIGHT IMAGE.

CYANOSIS RETINÆ.[1]

By Dr. HENRY H. TYSON, New York.

(With a drawing on Text-Plate V.)

IN 1861 Knapp first called attention to the changes in the fundi oculi known as cyanosis retinæ. Forty-four years later, in May, 1905, Posey read his paper on that subject before this society in which he referred to the literature, citing eighteen cases including his own with a brief résumé of each. Since then I have found no other recorded cases. Before reporting an additional case a brief review of the etiology and pathology of general cyanosis due to congenital cardiac disease as accepted at the present time and bearing upon the case may be of interest.

Various congenital cardiac diseases and malformations may cause fundus changes, viz.: "fœtal endocarditis, malformations resulting from imperfect development of certain parts usually septal, persistence of fœtal conditions, *e.g.*, foramen ovale or ductus arteriosus."

Patent foramen ovale or defect in the auricular septum, although one of the most common congenital cardiac malformations, about 50 per cent., is not one of the most important; it rarely occurs alone but is often found with pulmonic stenosis, to which it is frequently secondary, or it may result from imperfect development.

[1] Read before the American Ophthalmological Society at its annual meeting at New London, Conn., on July 16, 1908.

In pathological conditions the opening is from one fourth to one inch in diameter.

Pulmonic stenosis is one of the most frequent and important lesions. It may be due to fœtal endocarditis or to a malformation. It is more frequently associated with cyanosis than is any other congenital lesion. The amount of obstruction varies from a slight narrowing of the orifice to complete atresia.

While the symptoms of congenital cardiac disease are usually manifested soon after birth, they may not be noticed until after puberty. Cyanosis is the principal objective symptom and is present in over four-fifths of all the severe cases. It may be slight and only noticed upon exertion, or intense and constant, giving the skin a leaden color and the mucous membrane of the mouth a "raspberry hue." Holt states "that the view that cyanosis depends upon admixture of arterial and venous blood is generally discredited. In the great majority of the cases, at least, the explanation is a deficient oxidation of the blood in the lungs, owing to some interference with the pulmonary circulation. In 63 per cent. of the cases in his series of 242, there was found pulmonic stenosis or atresia of a small pulmonary artery. In cases with obstruction to the pulmonary circulation a polycythemia is present, the increase in the number of erythrocytes being proportionate to the cyanosis, the average being about 7,000,000; the hemoglobin is usually correspondingly increased, in one recorded case 140 per cent. The number of leucocytes is changed very slightly, disproving the theory of blood concentration."

Fromherz considers hyperglobulia and macrocythemia as oxygen carriers to offer a functional counterbalance for the cyanosis. Calabrese considers hyperglobulia to be due to irritation of the blood-producing organs by the overloading of the blood with carbonic acid gas.

The best explanation of the polycythemia seems to be that it is compensatory, and that the blood hypertrophies like other tissues, the blood-forming organs are stimulated to greater activity by the demands for oxygen. The quantity of the blood remains the same, but the number of erythrocytes and the hemoglobin increase and consequently the oxygen-carrying power is very greatly increased. This in part compensates for the smaller amount of blood that can traverse the lungs and there become oxygenated. (Holt.)

A condition similar to the cyanoderma of congenital heart affections occurs in the permanent hyperglobulia with splenic enlargement and cyanosis, but without heart lesion, which has been described by Vasquez. As there are congenital heart lesions with only very slightly pronounced physical signs, the differential diagnosis between the two conditions can only be made by the presence or absence of a disproportionately large spleen.

After congenital cyanosis has existed for a long time, structural changes are found in the veins which are dilated and have hypertrophied muscle in their walls. (Loubaud.) The capillaries become fibrosed and thickened, dilated and tortuous. The foregoing facts account for the prominence of the retinal vessels in these cases. Posey in his description of the vessels of the fundus in cases of cyanosis retinæ states that they attain a size which is rarely equalled in any other condition, this being particularly true of the veins, which may become enormously dilated and tortuous, the arteries, though usually similarly distended, being affected to a less degree, etc. I agree with him as to the veins being greatly dilated, but would state that in the fundi of some cases of dementia præcox I have observed veins equally as large, and in a few cases in the acute congestive stage, even larger, the relative size of veins to arteries being as four to one. This disproportion in size of veins to arteries being

one of the principal ocular signs of the disorder according to the Tyson-Clark syndrome.

The case of cyanosis retinæ which I am permitted to record came under my observation during the month of May, this year, and has the following history:

M. N.,[1] aged twelve years. Normal labor—was not a "blue baby" at birth; breast-fed up to nine months and apparently perfectly healthy. At about two years of age, during a convulsion, gastro-enteric in origin, he became blue, and this condition has persisted in varying degrees ever since. Has had measles, pertussis, and mumps; he has never been strong physically, and although he plays and runs with other children he tires quickly and gets very blue and very short of breath. He has severe palpitation after climbing stairs, and often complains of feeling cold especially in his hands and feet. Coughs upon exertion. Mentally he is very active and bright, but cannot concentrate his mind long at his books because his eyes tire and the print appears green to him, and he has headaches. He is very nervous and excitable but not as a rule irritable. Mental excitement produces very active cardiac palpitation. Appetite is very good and his digestion is normal. He has always been extremely constipated and is obliged to take cathartics daily. His chief complaint is dyspnœa upon exertion. *Physical Examination.*—Perhaps a little undersize for a boy of twelve years but well built and muscular. Skin has a bluish tint and mucous membranes are bluish black, as are also the tips of his fingers and toes. His heart seems slightly enlarged to right, action is of fair force and regular. Over the second and third spaces to the left of the sternum is a loud and harsh murmur with a musical element and apparently presystolic in time although it is continued throughout most of systole with a soft, blowing character. This murmur is transmitted up along the course of the pulmonary artery and over the second space is felt a slight thrill. No other murmurs. The lungs show congestion at their bases. Abdomen, both liver and spleen just palpable, otherwise normal; urine 1.012, acid—no albumin nor sugar.

[1] The patient was exhibited before the Ophthalmological Section of the New York Academy of Medicine at its meeting in May, 1908.

Excess indican. Microscopic examination negative. Blood from finger. Hemoglobin 120. White cells 9800. Red cells 8,080,000.

Dr. Walter James and Dr. L. H. Shearer, to the latter of whom I am indebted for the general history of the case, concurred in a diagnosis of congenital patent foramen ovale with pulmonary stenosis. (An idea of the cyanosis of his face which happened to be slight upon the day that his photograph was taken may be obtained by a view of the plates which Mr. Brulatour of the Lumière Process Co. kindly prepared for me.) The cyanosis showed itself in his ears, lips, mucous membrane of mouth, palpebral conjunctiva, and finger tips. He had clubbing of fingers and toes due to venous engorgement. The injection of the ocular conjunctiva was due to the dilatation of vessels, which also encroached on to the periphery of the cornea, the sensibility of which was diminished. Color fields were markedly contracted. Vision $\frac{20}{20}$. Exophoria 6° distance, 12° near. As will be noted by the drawing representing the fundus of the left eye (direct method), the fundus was much darker than normal with a violet tinge, disk appeared red due to dilatation of capillaries upon its surface, veins blue black, arteries usual color of veins, both being dilated and tortuous and extending to the periphery.

Nagel asserts that if the ophthalmoscopic examination reveals that the retinal arteries as well as the veins are darker and broader than normal the presence of a connection between the two sides of the heart is assured; whereas if the darker coloration and tortuosity is limited to the veins, he believes that the existence of some other congenital lesion is indicated, which does not permit of the admixture of venous with arterial blood. My case might be considered as a confirmation of this assertion, but before accepting it as a rule we must consider as a possible fact that we may have conditions in which we may get an admixture of arterial and venous blood in pulmonary diseases, *e.g.* emphysema and bronchiectasis. In emphysema, as Thomas points out, large areas of lung tissue have been absorbed, and it is easy to imagine that a

certain amount of blood may pass from the pulmonary artery direct to the pulmonary veins, without undergoing due oxygenation in the capillaries by the way. In bronchiectasis, again, areas of loss of substance occur, and tortuous, dilated capillaries with thickened walls exist, the blood-vessels here presenting a very similar appearance to that described by Carpenter in the lungs of a case of congenital cardiac disease. The organs were loaded with pigment, the capillaries dilated to three times their normal size, crowded with red cells, elongated, tortuous, their walls thickened and rich in young fibrous tissue elements. Must not many red cells have passed through these thickened channels without receiving their due share of oxygen and the blood have been thus returned, still largely venous in character, to the left heart?

Babinski's observation as to marked dilatation of retinal vessels preceding the cyanoderma in his case, I consider an important fact that should be a valuable aid in diagnosis in certain cases.

Treatment in these cases is of no avail and should be symptomatic. Prognosis, according to Vasquez and Quiserine, is fatally progressive when the polycythemia reaches 6,000,000 reds, evidencing more and more insufficient oxygenation and indicating a sudden change for the worse. As my case has a polycythemia of 8,080,000, the prognosis should be considered very unfavorable.

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2. Pflaundler and Schlossman. *The Diseases of Children* (Shaw and La Fétra), vol. iii., pp. 463–465.

3. Osler's *Modern Medicine*, chap. ix., pp. 323–342.

4. *Transactions American Ophth. Soc.*, vol. x., part iii., pp. 634–644.

AN IMPROVED METHOD OF BLEACHING PIGMENTED TISSUES.[1]

By Dr. F. H. VERHOEFF and Dr. CARL FISHER,
Boston.

THE method devised by Alfieri for bleaching sections[2] has two disadvantages: it is slow, and it does not bleach completely heavily pigmented tissues, such as the pars iridica retinæ. We have therefore endeavored to determine whether or not these defects could be eliminated. We have also studied, with bleached sections, the reactions of the more important staining methods after various fixatives, since this, apparently, has never been worked out. Sections of the eye were used because this process is of special interest to ophthalmic pathologists and because the pars iridica retinæ furnishes a convenient index of the progress of the depigmentation.

In Alfieri's method, the section is immersed in potassium permanganate, 1 : 2000, for 24 hours, preferably exposed to direct sunlight. The excess of permanganate is then washed out and the section placed in a solution of oxalic acid, 1 : 300, until the brown stain of the permanganate is removed, which requires about two hours. The permanganate solution does not keep well and has to be freshly made up to be effective.

The improvement in this method finally arrived at

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[1] From the Pathological Laboratory of the Massachusetts Charitable Eye and Ear Infirmary, Boston.

[2] *Monitore Zoologico Italiano*, viii., p. 57.

by us consists in greatly increasing the strength of the solutions. This not only reduces the time required from hours to minutes, but renders the method more efficient and the results more constant. Contrary to what might be expected, the sections are not made more brittle by these than by the weaker solutions, but rather less so. For either method it is, of course, important to have the sections thoroughly imbedded. Celloidin should be used for this purpose, since paraffin sections will not remain on the slide.

Of the various strengths tried, these were found to give the best results:

(1) Potassium permanganate, saturated aqueous solution (1: 16).

This should be made up with the aid of heat. It is found that the saturated solution will keep for weeks, if put in a dark place; a few days' exposure to the light, however, renders it much less effective.

(2) Oxalic acid, saturated aqueous solution (5%).

This also should be made up hot. Both solutions should, of course, be allowed to cool before using.

Sections are passed to the permanganate from water. For most specimens, 20 minutes in the permanganate will suffice; this bleaches even the pigment epithelium of the iris. Wash in water and place in oxalic acid until the brown stain of the permanganate is completely removed; 5 minutes is usually long enough. A quick examination in water, under the low power, will determine whether or not all of the pigment has been bleached. Wash in water and stain. If it is desired to bleach the pigment epithelium of the pars optica retinæ only, treatment in the permanganate for ten minutes will be found sufficient.

We have found that the best results are obtained after fixation in Zenker's fluid or formalin. After alcohol fixation, the nuclei stain poorly, while connective tissue stains diffusely, by the nuclear stains. Sections bleached by this rapid method have taken the various stains

uniformly as well as those bleached by the slower method of Alfieri, the resulting picture, however, being clearer on account of the more complete depigmentation. The following is a more specific statement of our findings in regard to the staining reactions of the bleached specimens.

Hematoxylin-eosin and Van Gieson's stain: These bring out the nuclei well and give the usual satisfactory general stain.

Marchi's method (for staining fatty degenerated myelin sheaths of nerve fibres): The black stain given the fatty elements by this method is not decolorized by prolonged bleaching.

Osmic acid: In sections stained by osmic acid alone, normal myelin may be afterwards bleached out, leaving the fat unaffected, so that a picture is produced similar to that given by the Marchi method.

Weigert's stain for elastic fibres: After alcohol fixation, this stains elastic tissue as usual, but connective tissue more deeply, thus obscuring the elastic fibres.

Verhoeff's elastic tissue stain (Jour. Amer. Med. Ass'n, March 14, 1908): This stains elastic tissue as well as before bleaching, but the protoplasm of smooth muscle stains diffusely, so that the arteries do not give the usual fine picture.

Una's orcein method for elastic tissue: After alcohol fixation, connective tissue in bleached specimens stains intensely, while the staining of the elastic fibres is impaired, so that the method is entirely unsatisfactory. To a less degree, this is true after formalin fixation. After fixation in Zenker's fluid, however, the reverse is the case; the staining of the elastic fibres is enhanced, while the connective tissue stains only slightly, giving a distinctly better picture than in the unbleached control specimens. The results, however, are inferior to those obtained with Verhoeff's stain.

Ziehl-Neelson method for tubercle bacilli: The bacilli stain more intensely in bleached sections.

The Gram-Weigert stain for bacteria in tissues: Previous bleaching does not affect the result.

Mallory's phosphotungstic acid hematoxylin (differential stain for neuroglia): This requires a slightly modified technique. Fixation in Zenker's fluid is essential. The sections should not remain in the permanganate more than five minutes, the proper interval being best determined by treating sections for 3, 4, and 5 minutes respectively; a difference of one minute here makes an appreciable difference in the result. After washing in water, the treatment with oxalic acid is omitted and the sections are passed directly to the phosphotungstic acid hematoxylin, where they are allowed to remain 24 hours. This gives a fine differentiation of neuroglia and is the best method for demonstrating the membrana fenestrata of the pigment epithelium of the retina.

Mallory's connective-tissue stain: Previous bleaching interferes with the action of the fuchsin, and hence this stain does not give a satisfactory picture, although the connective tissue stains as usual.

Weigert's stain for fibrin: This failed conspicuously in sections bleached 20 minutes.

Blood pigment and the usual extraneous pigments do not bleach by this method, so that it affords a simple means of differentiating them from the autochthonous pigments. The differential test for iron with potassium ferro- and ferri-cyanid may be made after bleaching, but the result is more striking if the iron test is made first and the section then bleached.

CONSANGUINITY A CAUSE OF CONGENITAL NYSTAGMUS.

By Dr. WILLIAM H. DUDLEY, Los Angeles, Cal.

INSTANCES in which nystagmus has been found to run through several generations in a family are not especially rare; and these instances often present various characteristics in the line of descent, and otherwise. The histories presented here are for the purpose of giving an instance in which consanguinity of marriage appears to be an important element in the transmission of the condition, for there appears to be but few instances in which heredity alone is sufficient to perpetuate the condition.

The patient who is responsible for this history is thirty-nine years old, female, and in usual good health; which latter also appears to be a family characteristic. In the case of the patient, ophthalmoscopic examination of the eyes is negative. Refraction: O.D. – .50 D. C. ax. H.; O.S. – .75 D. C. ax. H. With this correction, V. $\frac{20}{20}$ + with either eye. Nystagmus lateral, excursions moderate in degree and frequency.

The element of consanguinity of marriage for several generations has been so prominent that whenever various members of the family intermarry (which appears to have been rather frequently) "moving eyes" seems to have been expected in one or more of the descendants. The family are Germans, and appear to have been possessed of considerable wealth; and, according

to the statements of the patient, the frequent intermarrying was caused by a desire to "keep the money in the family." A study of the table presented, shows that on both paternal and maternal lines, in the four generations, there were forty-seven individuals, of whom twenty-six were nystagmic. On the father's side there were thirty individuals, of whom sixteen were nystagmic, while of the seventeen in the same number of generations on the mother's side, ten were nystagmic. As to sex: on the father's side there were seven males and nine females, on the mother's side there were four males and six females, nystagmic. Although this shows a larger number of females than males affected, the preponderance is not great. While consanguinity fills an important rôle in the histories, it does not appear essential in all cases; as in the case of the maternal grandfather, not a Ludwig,[1] and without the condition, yet he had four nystagmic children out of seven. It would be interesting to be able to present the degree of consanguinity in the various marriages recorded, but inasmuch as many of these occurred in Germany, and all are widely scattered geographically, this appears impracticable. While it is realized that the histories are incomplete, in not being able to follow out all of the descendants mentioned in the history, it is felt that enough has been presented to show in this case the influence of consanguinity of marriages, where a hereditary predisposition also exists.

Paternal grandmother—a Ludwig—"the only girl in the family"	Nystagmic
Paternal grandmother, 1 brother	Nystagmic
Paternal grandmother, 3 brothers	Without
Paternal grandfather—a Ludwig	Nystagmic
Paternal grandfather had 3 sons	Nystagmic
Paternal grandfather had 3 daughters	Nystagmic
Paternal grandfather had 2 others (sex unknown)	Without

[1] This name will answer here as well as any.

Paternal aunt, one of the 2 without nystagmus, married a Ludwig, 1st cousin:		
	Had 1 son	Nystagmic
	2 daughters	Nystagmic
	1 daughter highly myopic, and "turned head sidewise when looking"—conjugate deviation	Without
Paternal uncle one of those with nystagmus:		
	Had 1 son	Nystagmic
	1 daughter	Nystagmic
	1 (sex unknown)	Without
Father, one of the nystagmic males above referred to, married a Ludwig:		
	Had 1 daughter, the patient	Nystagmic
	3 sons	Without
	3 daughters	Without
Brother of patient, without nystagmus, married a Ludwig:		
	Had 1 daughter	Nystagmic
The patient did not marry a Ludwig:		
	Has 1 daughter	Without
Maternal grandmother—a Ludwig		Nystagmic
Maternal grandfather—not a Ludwig		Without
	Had 4 daughters	Nystagmic
	2 sons	Without
Maternal aunt married a Ludwig:		
	Had 3 sons	Nystagmic
	1 son	Without
Maternal aunt	Had 1 son	Nystagmic
	1 daughter	Nystagmic
Maternal aunt	Had 1 son	Nystagmic
	1 daughter	Without

CONTUSION OF THE EYEBALL, WITH SPECIAL REFERENCE TO TRAUMATIC ANIRIDIA AND APHAKIA.

By Dr. JULIUS FEJER, Budapest.

Abridged Translation from the German Edition, Vol. XLVIII., by Dr. Matthias Lanckton Foster.

CONTUSIONS of the eyeball are rather unusual because it is so well protected by the bony walls of the orbit, the bony framework of the nose, and the eyelids. This protection is least below, as the infraorbital margin is not very prominent. In contusions of the eyeball, the sclera is usually ruptured at a certain place above and parallel to the limbus. The reason that the sclera ruptures in this place is, according to Arlt's hypothesis, that the distention of the eyeball is greatest in the direction opposite to that of the blow and that the rupture always takes place at the apex of the distention. Other theories which have been advanced in explanation are that the eyeball is driven with such force against the bony margin of the orbit that the latter lacerates the former, and that the eyeball ruptures most easily in the place of least resistance, which is near the limbus. Mueller proved experimentally that a blow rarely strikes the eye from below, but usually from above and inward. He showed that the prominent bony margins were not only not always able to stop a blow, but might deflect it from its original course so as to enable it to hit the eyeball. Blows which come from directly in front and strike the

cornea never cause a rupture of the sclera because they drive the eyeball back upon the yielding orbital fat. Ruptures of the cornea are extremely rare. The etiology of those which occur is the same as that of scleral ruptures, and good recoveries are made in one half of the cases.

The most frequent result then of a contusion is a rupture of the sclera which is almost always parallel to the limbus. The iris may be partially or wholly forced out through this wound, frequently in association with the lens. When the conjunctiva is sufficiently yielding it does not tear, but the lens comes to lie partially or wholly in a pocket. Fuchs describes the case of a farmer who was struck in both eyes by the horns of an ox, each sclera ruptured, iris injured, and lens extruded, and after recovery saw very well with the aid of cataract lenses. Szili reported the case of a woman 56 years old, highly myopic in both eyes, who struck one eye against the projecting ornament of a chair. The sclera was ruptured, the iris and lens extruded. After removal of the prolapse the scleral wound quickly healed, and then the patient could see better with that eye than with the uninjured one. In another case reported by Szili a vertical linear wound of the cornea with ecchymoses of the lids and conjunctiva was produced by striking the eye against a projection in the dark. Recovery was complete in five weeks, but the iris had completely disappeared, while the lens remained in its normal position and was transparent except at a small spot to its inner side and above the equator. In the vitreous there were floating opacities and also a cobweb-like network, through a central opening in which the papilla was visible. The vision was $\frac{6}{36}$ with +4. In Businelli's case, after the blood in the anterior chamber had been absorbed the iris could not be found, but the lens retained its normal position, and the patient could with the aid of a stenopaic apparatus see as well as with the other eye. In

this case the iris was probably absorbed. Zander and Geissler collated eleven cases of injuries to the eye in which total aniridia had been produced. In Dixon's and Voss Salomon's cases, the iris and lens were extruded through a rupture in the sclera, but after recovery the patients saw well with convex glasses. In Praun's case, a wound *6mm* long was produced. After recovery the iris was absent, and the patient had 0.7 vision with a convex cylindrical lens. In Lyder Borthen's case there was a rupture of the sclera and absence of iris and lens, but good vision was obtained. In Praun's second case, a boy struck his father in the eye with his finger and caused a hemorrhage in the vitreous. After the blood had been absorbed, the iris and lens were found to have been totally detached. Shreds floated about in the eye and became absorbed. No laceration of the sclera could be detected although the lens could not be found. With correction the patient had $\frac{6}{60}$ vision and could read Sn. 1. In Tvyl's case total aniridia and aphakia were produced without demonstrable laceration of the outer coat of the eye. After the blood had been absorbed and the vitreous had cleared up, a shrivelled gray object surrounded by pigment could be seen in the region of the ciliary body and was supposed to be the remains of the iris. It was also supposed that the lens had become completely absorbed as the result of a rupture of the capsule. The vision was $\frac{6}{36}$ with +10 D.

The exciting causes mentioned in literature are, in the order of frequency, blows with the fist, blows with the fingers, impacts on prominences of various objects, and a blow with a bar of iron.

In the case which I am about to relate, the iris and lens were torn away without extrusion from the eye through a rupture of the sclera. They became absorbed and left almost no traces and the patient obtained satisfactory vision with a correcting glass. It should be stated that the patient consulted another ophthalmologist

on the second day after the receipt of the injury and came to me on the eighth day, but no trace of scleral rupture was found by either of us. Tvyl's case is the only similar one reported, as in Praun's scleral rupture could not be excluded with certainty.

A woman, 48 years old, slipped and fell Nov. 18, 1901, striking her right eye against a projecting knob on a chair. The eye immediately became blind, but not very painful. On Dec. 11, 22 days after the injury, there was marked ciliary injection, no injury of the sclera to be found, a very deep anterior chamber, and absence of iris and of the reflexes of the lens. The ciliary processes were invisible. Deep in the eye could be seen a solid body with fringed edges, possibly a portion of the iris or the lens, or both. It could not be the retina, because the projection of light was good. Vision was movements of the hand. The tension of the eye was not increased. Treatment consisted of warm applications and potassic iodide internally to promote the absorption of the blood. It took several months for the vitreous to clear up, but finally there could be seen in the inner-lower portion a membrane with irregular margins floating about. Deep in the eye was a gray mass, and in addition there were many floating opacities in the vitreous. On June 16, 1902, the vision was $\frac{5}{10}$? with + 15 D. The condition of the eye continued to improve and the fluttering membrane atrophied until the entire fundus was visible. On Nov. 26, 1902, the vessels of the papilla and retina showed no abnormality, a body of uncertain size and indistinct margins could be seen by oblique illumination in the median line in front of the equator, probably the remains of the lens, and the absence of Purkinje's images together with the patient's vision of $\frac{5}{10}$ with +19 D. demonstrated that the lens was not in its normal position. The visual field was contracted upward and inward to 40, downward and inward to 45, upward and outward to 40, and downward and outward to 20. Letters could be seen more clearly with the aid of a stenopaic slit. At this time the fundus, or at least a portion of it, could be seen without the assistance of the ophthalmoscope or of a lens when the patient was so placed in a lighted room that the

light fell laterally on the eye and the observer looked into the eye from the opposite side. This was due to the fact that the absence of the iris not only allowed much light to enter but also to be reflected out from the eye, and in consequence of the aphakia and high hypermetropia the reflected rays united at a certain distance to form a distinct image.

Now what became of the iris and of the lens? As the sclera did not rupture, they could not escape from the eye. As already mentioned, blows which fall on the cornea directly in front never rupture the sclera, because they press the eye back upon the elastic orbital fat. In this case the patient struck her eye against a projecting knob on a chair and the anterior segment of the eyeball, the cornea, may have been driven into the posterior, so that the force of the impact was transmitted directly to the iris and lens. I take it that the force of the blow was divided equally throughout the anterior segment of the eye, and therefore that the prominence on the chair had a smaller diameter than the eyeball, because only with a smaller sphere can a larger one be symmetrically indented. Under such conditions the iris has usually been found as a gray shrivelled mass in the anterior chamber, although Delacroix claimed that it might be wholly absorbed. Praun accepted that theory, but Gayet and Lyder Borthen asserted that a shrivelled portion of the iris must always remain. In this case a fluttering membrane with ragged edges was seen to gradually undergo absorption until only a trace of pigment remained. Was the lens absorbed, or did it lie somewhere on the ciliary body where it could not be seen with the ophthalmoscope? The latter hypothesis is hardly possible, because on account of the total aniridia almost the entire cavity of the globe could be investigated. Experience on the other hand is very favorable to the possibility of its absorption. In young people the lens disappears leaving no trace in the course of a few weeks after discission of the capsule, and in elderly people the

cortical matter left behind after extraction of a cataract undergoes rapid absorption. Many cases have been reported in which an over-ripe cataract has shrunken so that the pupil became black and the patient was able to see. Spontaneous absorption is not so rare. Szili reported a case in which a cataract which had been pronounced ripe three and a half years before had shrunk so much and separated from the zonula so fortunately that the patient regained his vision. Reuss in 1900 collated 34 cases in which senile over-ripe cataracts in unwounded capsules were absorbed and disappeared from the region of the pupil. Hence I may assume that in this case also the lens became absorbed after it had been luxated and the capsule lacerated by the contusion, and had become opaque through the entrance of aqueous, or of blood, associated with imperfect nutrition. The gray body which finally came to light in the depths of the eye was probably the remains of the capsule.

In conclusion I wish to mention a condition observed and described by Dixon. The sclera about the cornea was flattened and drawn in so that the eyeball had a conical form. The reason of this could not be determined with certainty, but it was thought to be probably dependent on the total detachment of the iris and the traction of the ciliary processes. According to Praun, in aniridia the eyeball forms a complete cavity, and the pressure of the extrinsic muscles together with the altered conditions of tension produces a flattening or drawing in of the region of the limbus. In my case this condition was not present—the eyeball retained its normal form.

PIGMENTATION OF THE RETINA FROM THE VITREOUS CHAMBER. TYPICAL RETINITIS PIGMENTOSA.

By Dr. ERNST V. KNAPE, Finland.

Translated from the German edition, Vol. XLVIII., by Dr. Matthias Lanckton Foster.

OUR knowledge in regard to the etiology and nature of retinal pigmentation is as yet so faulty that any contribution toward the determination of these subjects is of interest.

The eye in this case came from the practice of Prof. Schweigger. The patient was an elderly man, myopic, who had suffered from a detachment of the retina which by degrees had become total. After this condition had persisted for some years inflammatory symptoms manifested themselves. The most noteworthy point was that fine pigmentations became visible with the ophthalmoscope on the detached retina. A chronic iridocyclitis soon afterward cut off the view of the interior of the eye and finally the blind and painful organ was enucleated.

The eye was divided at the equator. After the fairly clear fluid remains of the vitreous had escaped the fine pigmentation could again be seen everywhere on the detached retina. The posterior segment alone of the globe was preserved. It was placed in a 10% formalin solution so as to preserve the transparency of the retina, which was totally detached except at the papilla. Beneath the retina was a slightly milky coagulated exudate.

Even with the naked eye coarse dark spots and stripes could be seen on the retina. The retina was attached to the choroid at only one place which was about the size of a pin's head. Examination with the binocular loupe showed dark round spots and branching dark slender lines on the retina. The choroid was pale, poor in pigment except at the place where it was attached to the retina. At that point it was densely pigmented.

I set apart a small piece of the retina for teasing, embedded in paraffin a second, which included the place where it was attached to the choroid, stained a third piece with carmine, and embedded the balance in celloidin. Under the microscope the specimen stained with carmine showed the same characteristics as under the binocular loupe, except that it was evident in this as well as the teased specimen that the dark places were formed by free pigment granules and pigmented spots. Later on I will describe this pigment.

Microscopic Examination of the Paraffin and Celloidin Sections.

The sclera at the posterior pole was thin, as is commonly the case in myopia, but otherwise normal, but a very interesting condition was presented at the places where the ciliary vessels pass through the sclera. The ciliary vessels were sheathed in pigment at these places in a manner similiar to that in which the retinal vessels are often sheathed in retinitis pigmentosa. The pigment had doubtless migrated from the choroid into the lymph spaces and connective tissue about the ciliary vessels and nerves where it lay in irregular lumps and not enclosed in cells.

The choroid was greatly changed. Even at the first glance it was evidently very atrophic. A closer examination showed that the vessels had been affected first of all. Only a few scattered capillaries remained of the

chorio-capillaris, some of the medium-sized vessels remain, but they were fewer in number than normal, and a considerable portion of both these and the large vessels exhibited characteristic changes.

The easiest way in which the pathological process may be traced is to begin with a large vessel. The intima of such a vessel was found to be thickened, often folded, so as to reduce its lumen, while newly formed elastic lamellæ could be seen. Even the adventitia was thickened and the vessel was surrounded by newly formed connective tissue. I am in doubt whether the media took part in the pathological process or not. The medium-sized vessels were often so narrowed that only an inconsiderable lumen was left. This pathological process of the vessels was inflammatory beyond a doubt and it agreed in every respect with endarteritis obliterans. We will return to this later.

The pigment epithelium was greatly degenerated. The different stages of degeneration could be studied best in the sections which had been made obliquely through the choroid. There were normal hexagonal cells with uniform distribution of pigment. In commencing degeneration the fuscine granules had gathered together and the distribution within the cells had become irregular. Later the hexagonal form of the cells had become round, or elongated, and the cells themselves appeared to be swollen, while a part of their walls had disappeared and the fuscine granules had escaped from them. Frequently the cells had become loosened, had assumed a round form, and had migrated into the retina. The pigment epithelium was entirely gone over large spaces. The stroma pigment in the choroid was rarefied. Here and there a cell was to be seen with the pigment granules escaping, but the changes were more difficult to see than those of the pigment epithelium.

In the retina the rods and cones had disappeared except at a few places where isolated deformed ones

were to be seen. The external limiting membrane was destroyed in some places, well preserved in others. The other layers were much deformed and merged irregularly with one another, the internal granular being the best preserved. Ganglion cells were scanty and for the most part strongly pigmented. The internal limiting membrane was destroyed in places. There was a slight proliferation of the supporting tissue of the retina. The vessels were greatly narrowed by hyaline degeneration of their walls. Some leucocytes were present in the tissue. Within the retinal tissue there were numerous spaces, some long and irregular, others round and fairly regular.

The pigmentation of the retina was of the greatest interest. Even on a superficial examination it was evident that this was of two kinds, viz:

(*a*) Black pigment granules, some separate, others formed into large lumps, on the internal limiting membrane. These were never intracellular, but were lying free on the inner surface of the membrane. As has already been mentioned, this membrane was destroyed in places, and at those places the black granules passed into the retinal tissue where they could always be distinguished very clearly from the other kind of pigment.

(*b*) A migration of pigment from the degenerated pigment epithelium described above into the layer of the retina next to the choroid. This pigment consisted partly of brown granules larger than those described under (*a*), partly lying free in the retinal tissue, but for the most part enclosed in cells which were more or less deformed migrated pigment epithelia. These cells were most numerous in the outer layers of the retina, but were not rare in the inner layers.

In the perivascular lymph spaces, or the connective tissue about the vessels, were pigment cells and accumulations of free pigment granules. It was difficult to determine with certainty to which kind the latter be-

longed, but they seemed to belong to both. No pigment was found within the blood-vessels.

It has been mentioned that in one place the retina was adherent to the choroid. That place exhibited under the microscope a marked proliferation of connective tissue, which enclosed numerous cavities, which were often lined with newly formed pigment epithelium. At this place the retina was penetrated by connective tissue. Of its different layers a granular alone could be recognized here and there, but which one could not be determined.

Both the clinical and the pathological-anatomical pictures are those of retinitis pigmentosa. The only deviation is that the pigmentation appeared after the retina had become detached. But this is not inconceivable because Profs. Greeff and Brückmann have shown that broken-off pigment epithelial cells may migrate into a previously detached and atrophic retina, as for example in the so-called pseudo-glioma. Or was the pigment carried thus by swollen leucocytes? In this case the pigmentation of the inner surface of the retina was peculiar and was doubtless due to a migration from the vitreous chamber. There are only two similar cases on record.

Schweigger mentions a case of iritis, peculiar changes in the region of the ciliary body, excavation of the disk, and atrophy of the nervous retinal elements, with independent development in the retina of black masses of pigment about the retinal vessels, not enclosed in cells. The choroidal epithelium was normal. Later in his *Handbook of the Special Diseases of the Eye* he claims that it is possible that the pigmentation came from the pathologically changed epithelia of the ciliary body.

The other case was described by von Hippel, who enucleated the eye on account of glaucoma. In the trabecular tissue which separated the anterior chamber from Schlemm's plexus, as well as in the iris and on its anterior surface, there were numerous pigmented cells

The pigment epithelium of the ciliary body had lost much of its pigment. There was no penetration of pigment into the retina from outside except at a point in its most anterior portion, immediately in front of its transition into the pars ciliaris retinæ, where a small mass of pigment cells did thus penetrate. But on the inner surface of the retina there were little clusters of pigmented cells which in places penetrated the retina and lay about the vessels. Such cells were also present in the vitreous. Von Hippel was of the opinion that this pigmentation came from the ciliary body.

The pigmentation of the retina in these cases came from migration of pigment from the ciliary body through the vitreous. Unfortunately I was unable to examine the anterior portion of the eyeball in my case, but I may assume that here also the pigment on the inner surface of the retina came from the anterior part of the uvea. It is not clear to me whether the extension was active or passive. I did not find the pigment to be intracellular, and this condition seems to indicate that the extension was passive.

Etiology of Typical Retinitis Pigmentosa.

In reading the literature on the subject of retinitis pigmentosa I was struck by the fact that complications were present in most of the published cases, and the thought occurred to me that there might be a causal relationship between these complications, developmental anomalies, and the disease. I think I have discovered such a causal relationship and present the following in the hope that I may be able to throw some light upon the etiology of this disease.

It has been noticed for a long time that in persons suffering from retinitis pigmentosa such developmental anomalies were present as deafness, harelip, polydactylia, persistent hyaloid artery, posterior cataract, coloboma

of the iris or of the choroid, microphthalmos, faulty development of an entire limb or of a part, congenital atrophy of the optic nerve, etc., as well as neuro- and psycho-pathological conditions. It is easy to infer, as was pointed out by Siegheim, that all persons in whom this disease develops have developmental anomalies of a part of the eye which predispose them to retinitis pigmentosa. Some authors have considered consanguinity of the parents or ancestors responsible for these arrests of development; others consider heredity, others syphilis, to be the chief factor. It is unnecessary for us to enter into this contest. It is sufficient to say that consanguinity is a cause of this developmental anomaly and that heredity transmits it to posterity. I will only say here that children of perfectly healthy parents may present these anomalies. Regarding this Ziegler says: "If such anomalous characteristics frequently appear in children of parents who are themselves normal and are of healthy ancestry we must suppose that the sexual seeds of the parents were of themselves normal, but by their union produced a pathological variation."

Taking into account what has been said above, I advance the view that the persons in whom retinitis pigmentosa develops are afflicted by a degenerative anomaly of development dependent on a peculiar pathological germ variation which predisposes to the production of this disease.

Now as to the kind of this anomaly. The structure of the eye and the pathological examinations give us a suggestion.

Even in 1859 Schweigger claimed that the presence of pigment had an adventitious signification, and later writers confirmed his opinion.

Most authors who have investigated cases of retinitis pigmentosa pathologico-anatomically, like Wagenmann, who by his experiments established the importance of the choroid in the nutrition of the retina, think

that the kind of disease should be sought in changes in the perceptive elements of the retina caused by disturbances of nutrition, which in turn are dependent on changes in the choroidal blood-vessels. My own investigation shows the presence of endarteritis obliterans in the choroidal vessels. Bearing this in mind, I may assume that the choroidal vessels in any individual who has a congenital tendency to retinitis pigmentosa form a place of less resistance, and that this anomaly of development predisposes to endarteritis obliterans. But granted this predisposition there is still need of an exciting cause. If we closely examine the recorded clinical histories of retinitis pigmentosa, we find in most of them complications, some of them disregarded, some taken to be the cause of the disease, and in them I look for the exciting cause. My assertion cannot be gainsaid by the fact that we cannot demonstrate this cause in all cases, because a slight choroiditis anterior may be easily overlooked in childhood, or forgotten later. I will adduce some facts taken from literature,

The eye examined by Donders had been blinded by variola. H. Mueller described a case with concretions in the papilla. One of Junge's cases was complicated by posterior ectasia; in the other, one eye had been blind from birth. One of Schweigger's cases was associated with hydrophthalmos, deep excavation of the optic nerve, and chronic iritis with occlusion of the pupil; in another there was amaurosis after reclination, with cicatricial contraction of the cornea and adherence of the iris; a third was complicated by iritis, peculiar changes in the region of the ciliary body, and cupping of the nerve. In a considerable number of the remaining cases which were examined pathologico-anatomically and reported, the clinical histories are faulty.

I can add further examples from exclusively clinical observations. In the following table I present a com-

parison according to Schmidt, with additions from Schön and Herrlinger.

		Author										
		Leber	Denti	Bayer	Nolden	Derigs	Vider	Siegheim	Schmidt	Ancke	Schön	Herrlinger
No. of cases of retinitis pigmentosa		66	10	19	14	27	42	73	43	7	6	92
Complications	Syphilis	5[1]	1	0	0	0	..	2	2	..	..	2
	Acute febrile diseases	..	..	..	..	3	7	6	2	1	..	17
	Complications about the eyes	..	..	7	10	15	4	19	18	5	6	48

It is to be noticed that the histories are as faulty in this table as in most reported cases, particularly as regards syphilis and the acute diseases of childhood. The acute fevers included in the table were typhus, scarlatina, measles, erysipelas, pertussis, and meningitis. Influenza and variola are also mentioned in reported cases. The ocular complications were extremely variable and some certainly began after the commencement of the retinitis pigmentosa. These imperfect histories preclude deeper penetration into the subject. Other complications seem certainly to be in close relation to the retinitis pigmentosa. Schön's cases are brilliant examples. All the six patients were children, the disease was still in its early stage, and a careful examination revealed choroiditis anterior in each one.

Various diseases have been charged with the responsibility of being the cause of retinitis pigmentosa, such as malaria, rheumatism, epilepsy, syphilis, pellagra, and dyscrasiæ of all kinds. It has also been ascribed to traumatism and to dazzling. In this connection I will quote from Herrlinger's *Inaugural Dissertation:* "But what reliance can be placed on the data given by the patients is shown by Case 7, of Marie E., who stated that

[1] Syphilis suspected.

prior to an attack of measles in her thirteenth year she had had good vision and that her eye trouble dated from that time. According to Magnus and Siegheim, the measles would have to be considered the excitant to development (Entfaltungsreiz), although the disease was beyond doubt congenital, as her brother had retinitis pigmentosa without a preceding attack of measles. If this patient had happened to have no brother, the disease would certainly have been thought to have resulted from the measles." This case may be explained without reference to the brother if we suppose the measles to have been the exciting cause of the retinitis pigmentosa, and I agree with those writers who believe in such an exciting cause.

The question now arises how the infectious disease retinitis pigmentosa can be called forth, and I will try to make clear my opinion in this regard.

Any infectious disease may affect the eye in different ways. Microbes or toxins may be carried by the circulation directly into that organ, or the eye may be affected by a pathological condition of the central nervous system occasioned by the infectious disease, or thromboses may be formed in the vessels, or the vascular power of resistance may be impaired in consequence of the reduced general strength. In our particular case I think that the acute infectious disease caused an inflammation of the chorio-capillaris, which in this individual was a locus minoris resistentiæ. It is not a matter of surprise to us that this should be the case when we remember that infectious diseases are often complicated by choroiditis. This inflammation of the chorio-capillaris then produced the endarteritis obliterans.

Among the infectious diseases which have had inflammatory conditions of the uvea associated with them are variola, varicella, influenza, cholera, typhus, typhoid, relapsing fever, septicæmia, pyæmia, acute articular rheumatism, malaria, and mumps. The relation of

syphilis to retinitis pigmentosa is a mooted question. While some authors acknowledge no connection between the two diseases, others consider retinitis pigmentosa as a syphilitic disease, and many claim simply that sometimes syphilis plays a part in its etiology. I belong to the third group and regard syphilis as a common exciting agent because of its general prevalence and of its tendency to reduce the vital resistance and particularly to affect the vessels.

In many cases the differential diagnosis between retinitis pigmentosa and retinitis syphilitica is very difficult if not impossible. Such would seem to be cases in which a retinitis pigmentosa is set up associated with syphilitic changes. Thus Leber says in *Graefe-Saemisch's Handbook* that in no case in which syphilis was the probable cause were all the symptoms of typical pigment degeneration present, while the onset and course were frequently anomalous.

Sidler-Huguenin in his recent work describes four different types of fundus affections due to hereditary syphilis. Regarding Type iv. he says that we have to deal here with a clinical picture which closely resembles that of retinitis pigmentosa. It is a neuro-chorio-retinitis, or, stated more correctly, a chorio-retinitis with secondary pigment degeneration and optic atrophy. This fundus disease occurs relatively seldom in hereditary syphilis and is often mistaken for typical pigment degeneration on account of the great similarity between the ophthalmoscopic pictures and the functional disturbances. In each of the twenty-one cases quoted by the author both eyes were symmetrically affected. The course of the disease is always tedious. He further says that it may be concluded that the patients of Type iv. have in addition to the true symptoms of hereditary syphilis a special degeneration which is analogous to that of retinitis pigmentosa, but he does not state what kind of a degeneration this may be. This difficulty is done away with,

however, if we say, on the grounds stated above, that the patients of Type iv. have, in addition to the true symptoms of hereditary syphilis, a special degeneration which is identical with retinitis pigmentosa. The explanation is that these patients have a congenital predisposition of the choroidal vessels toward endarteritis obliterans and the syphilis excites a retinitis pigmentosa. This theory of mine is confirmed by the observations of this author that in this type there are met with in addition to the symptoms of hereditary syphilis certain signs of degeneration which resemble the anomalies which frequently accompany retinitis pigmentosa.

Sidler-Huguenin further says that the prognosis in Type iv. is better than in retinitis pigmentosa. None of his twenty-one cases was blind, eleven were between the ages of 20 and 38, the rest were younger, and none had been incapacitated for the performance of their daily duties. This point is of no differential diagnostic value and does not exclude the presence of retinitis pigmentosa, because the central vision is usually lost late in the latter disease,—according to Fuchs in or after the fiftieth year. When I consider this I am only strengthened in my view that Sidler-Huguenin's Type iv. includes patients with congenital predisposition in whom syphilis has excited a retinitis pigmentosa.

This congenital predisposition may result in retinitis pigmentosa at any age, although naturally the disease appears most frequently during childhood. It may commence even during intra-uterine life, and this is not surprising when we remember that children are born with syphilitic changes. On the other hand, the abnormal condition of the choroidal vessels may remain latent for many years. Hutchinson mentions two cases in which the disease first appeared during the 18th year, and Bürstenbinder reports a case in which it came on during the 56th year. I think that there are also cases which live under such good conditions that the

disease never breaks out, but yet those persons may transmit the anomaly of development to later generations. This would be similar to the hereditary relations of hæmophilia and in agreement with indirect, latent heredity.

In conclusion I will briefly summarize my theory. The cause of retinitis pigmentosa is twofold: a predisposition on the part of the choroidal vessels to endarteritis obliterans due to a congenital anomaly, and an excitant in the form of some disease of the eye, a fever, or syphilis. The nature of the disease is a gradually progressive endarteritis obliterans which begins in the smallest capillaries of the choroid behind the ciliary body. As a consequence of altered nutritive conditions, this process produces an atrophy of the retina which slowly proceeds from the periphery toward the centre, together with a secondary migration of pigment into the retina.

There are several points in this explanation of the etiology of retinitis pigmentosa which are not clear, but I am convinced that accurate observations of the data given by the patients will elucidate these points in the future. It would be particularly of interest to determine whether the patients are affected with endarteritis of other parts of the body besides the choroid.

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HERPES ZOSTER OPHTHALMICUS AND PARALYSIS OF THE TROCHLEARIS.

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Translated from the German Edition, Vol. XLVIII., by Dr. Matthias Lanckton Foster.

IN spite of the amount of literature on the subject, the nature of herpes zoster is but little understood. If we accept as a well established fact that it is an inflammatory affection of the sensory nerves, the cause of the characteristic cutaneous eruption is still wholly unknown to us. The theory that it is an inflammation of the sensory nerves has been defended recently by Wilbrand and Sänger, who hold that in herpes zoster we have to do with a neuritic process dependent on a specific infection which often draws into sympathy the corresponding spinal ganglia. This theory is supported by the fact that paralyses of motor nerves occur in association with herpes zoster. In this respect the ophthalmic variety holds a prominent position, because it is as complications of this form that paralyses of the ocular muscles are most frequently mentioned in literature. Yet the actual number of such observations is small. The paralysis most frequently mentioned is the partial of the oculomotorius, the next the total of the same muscle. The abducens is more rarely involved and there is almost no mention in literature of the involvement of the trochlearis. Wilbrand and Sänger quote a case from Lesser, and a case was reported by Ginsberg in which there seemed

to be a paralysis of the trochlearis associated with a partial paralysis of the oculomotorius. According to Wilbrand and Sänger, the occurrence of facial paralysis is very exceptional with herpes zoster ophthalmicus, but more frequent with herpes zoster occipitalis. Hence the report of a case in which motor paralyses occurred in conjunction with herpes zoster ophthalmicus is not without interest.

In the following case there was a total isolated paralysis of the trochlearis coincidentally with a herpes zoster ophthalmicus and a total facial paralysis of the opposite side after the cutaneous affection had passed away.

J. K., 69 years old, was attacked early in October, 1902, by a slight chill and other febrile symptoms, together with a painful roseate eruption on the right temple. The redness extended from the middle line to in front of the right ear, upward high in the parietal region and downward to the zygoma, covering the lids, which remained swollen for a long time, and the right side of the nose almost to the alæ. In many places on the brow and temple, the scalp, the upper lid, and about the inner canthus pustules appeared, dried up, and healed, leaving depressed cicatrices. About the third or fifth of November the patient noticed diplopia on looking downward. It was at that time that he was first able to open his eye spontaneously on account of the disappearance of the inflammatory swelling. The eyeball had been slightly reddened throughout.

Nov. 19, 1902.—General condition very good, nutrition normal. Patient complains of nothing except the diplopia. On the right side of the brow, extending as far back as the parietal region, there were perhaps twenty irregular, small, deep, brownish-red cicatrices in the skin, and some very small ones of the same characteristic appearance on the right side of the nose, but none on the temple or on the lids. Elsewhere the skin was of normal color. Sensation was slightly lessened on the right side of the brow, where a touch was felt less than on the left side and the prick of a needle caused scarcely any pain. In the right temporal region,

as low down as the zygoma, deep punctures with a needle were unperceived. The skin of the lids, the conjunctiva, and the cornea retained their normal sensibility. There was no sensory disturbance on the left side of the face. There was no disturbance of the motility of the facial muscles on either side. Both eyes were externally normal, the corneæ perfectly clear, the pupils small and normal in reaction. The right optic disk was perhaps a little pale. The refraction was myopic. R. V., $\frac{1}{2}$ with —1.5 D.; L.V., almost 1 with —1 D. The accommodation of each eye corresponded to the age.

No fault in the motility of the eyes could be detected by external examination, and it was necessary to use a red glass and a candle in order to ascertain the cause of the very annoying diplopia. The results obtained were: above, single vision; on the horizontal meridian to each side as well as directly in front a yellow light was seen directly below the red light seen with the left eye, which became fused when the eyes were turned slightly upward; below, there were homonymous double images, the one belonging to the right eye deeper and distinctly nearer. The distance between them was least downward and to the right where it was perhaps a hand's breadth. At this place an obliquity was clearly recognized, the point of the lower image inclined toward the upper. This last statement was made spontaneously by the patient and, so far as I can remember, it was the first time I have known such a statement to be volunteered by a patient undergoing examination for paralysis of the trochlearis. If the candle was carried from the right toward the middle line the distance between the images increased, and if carried over to the left it was stated to be at least one metre, the upper image to have appeared to come noticeably nearer, and the obliquity to have disappeared.

This picture is one of an uncomplicated paralysis of the right trochlearis with a diplopia so typical, thanks to the statements of the very intelligent patient, that

the case would satisfy any teacher as an object for demonstration.

Potassic iodide was prescribed and the right eye ordered covered.

On Nov. 30th the patient presented himself, to my surprise, with a total paralysis of the left side of his face. According to his very precise statement, this paralysis developed within a very short time during the forenoon of the 26th, without known cause and without premonitory symptoms.

The left side of the brow was smooth and could not be wrinkled. The palpebral fissure was wide, the lower lid hung down and allowed the tears to run over it. When the patient tried to squeeze his lids together they approached until about 5*mm* apart. The eye could not be closed. On looking downward the upper lid moved equally as well as the other. The left cheek was relaxed and sunken in, the mouth drawn over to the right with its left angle somewhat drooping. Whistling was impossible. The left side of the velum palati hung somewhat lower than the right, but moved well in intoning. The tongue was protruded straight out. There were no sensory disturbances on the left side, no changes in the limbs, and the general health was undisturbed. The paralysis in the right eye had improved somewhat. The patient carried his head inclined forward and turned to the left.

To the treatment with potassic iodide was now added daily peripheral galvanization of the facial nerve, and the cathode was applied for several minutes to the region of the tendon of the right trochlearis. Although at first cathodal and anodal closure contractions could be obtained only in the muscles of the brow, the galvanic excitability improved pretty rapidly, hand in hand with the return of the spontaneous mobility, first in the portions of the nerve about the brow and eye and lastly about the mouth. On Dec. 7th the left side of the brow could be wrinkled

almost normally, and with some exertion the patient could very nearly close the eye. Although the nerve did not appear electrically responsive about the mouth, the patient noticed an improvement as he could drink and smoke with greater ease. On Dec. 10th the tears still overflowed the left eye, but the closure of the lids was almost unimpeded, even without special exertion, and the function of the facial about the mouth had improved to such an extent that the patient could whistle. At this time the first closure contractions of the left depressor labii inferioris appeared. On Dec. 17th the left palpebral fissure was still somewhat wider than the right. The lower lid stood a little out from the eye and the tears overflowed when the eye was exposed to the wind, but the lids could be closed easily. The left cheek and region of the mouth were still somewhat relaxed, the electric reaction nearly normal everywhere. At the last examination, Jan. 10, 1903, the left side of the face seemed to be sound except that the left angle of the mouth was somewhat less innervated. The electric reactions showed no further change.

On the right side the anæsthetic area had steadily contracted. On Dec. 17th, although sensation was elsewhere normal, the prick of a needle was unperceived only in a region a finger's breadth above the zygoma and half as far back of the temporal end of the eyebrow.

The paralysis of the trochlearis had also rapidly receded. On Dec. 10th the double images were separated to the right and downward by a distance of three fingers' breadth, to the left and below by a hand's breadth. On Dec. 17th these distances were two and four fingers' breadth respectively. The diplopia began now just below the horizontal meridian on the left side. Subjectively the patient was scarcely troubled by it even when the right eye was uncovered, because he could compensate for the disturbance by the position of the head. On Jan. 10, 1903, all traces of the paralysis had disappeared.

In this case there was an attack of typical herpes zoster ophthalmicus or frontalis with symptoms of a general infection accompanied by fever. Four weeks after the beginning of this disease, or perhaps a little sooner, an isolated, total paralysis of the trochlearis of the same side of the face appeared, and three additional weeks later there was a sudden attack of total paralysis of the facial of the opposite side. At an early period there was a noticeable involvement of the second branch of the trigeminus. All paralyses have a marked tendency to recovery, which seems to be peculiar to motor disturbances which result from herpes zoster, and this progresses the more rapidly the farther removed the symptoms are from the fundamental lesion.

As to the causal connection between the partial paralysis of the second branch of the fifth nerve and of the trochlearis with the attack of herpes zoster in the region of the first branch of the fifth nerve it can scarcely be questioned. But it is otherwise with the facial paralysis on the opposite side, which may perhaps be taken for an accidental complication. But the best explanation of the entire clinical picture is obtained if it is assumed that there was an acute infectious neuritis or perineuritis which attacked first the first branch of the fifth nerve and the Gasserian ganglion, then the second branch of the fifth and the trochlearis, which immediately adjoins it in the middle fossa of the skull, and finally sprang over to the facial nerve of the opposite side. Even if the paralysis of the latter, which appeared long after the other symptoms and without any febrile symptoms, is to be considered a distinct affection not etiologically connected with the preceding herpes zoster, still the clinical picture furnished is very interesting. The coincidence of a total paralysis of a motor nerve, the trochlearis, running so close to the trigeminus affected with herpes zoster ophthalmicus, can certainly serve as a basis for the theory of a neuritis in this case, and

the inference would be very plausible that the same inflammatory changes involved, by way of the Gasserian ganglion, the first and second branches of the fifth and then the fourth nerve.

A RARE CASE OF PERSISTENT PUPILLARY MEMBRANE.

By Dr. K. RUMSCHEWITSCH, Kief.

Translated from the German Edition, Vol. XLVI., by Dr. Matthias Lanckton Foster.

IN my article on the "Anatomy of the So-called Persistent Pupillary Membrane" (*Arch. f. Augenh.*, xx., p. 314) I gave the results I had obtained from the anatomical study of this anomaly. By these and by the results obtained by others I was led to the view that the "persistence" of the pupillary membrane should be looked upon as a consequence of the abnormal development of the membrane during embryonal life. If in these cases the membrane followed the type of the iris in its development it would be unable to atrophy in the manner of the normal embryonal pupillary membrane. Hence I think that we have to do not with a persistence, but with an abnormal development of this membrane. This condition is not particularly rare, as more than two hundred such cases are recorded. It has been observed in very different forms as membranes or fibres which started from the anterior surface of the iris and formed a sort of incomplete diaphragm between the anterior and posterior chambers, or were attached to the anterior capsule. Exceptional cases are found in literature. Beck (Ammon's *Zeitschrift*, Bd. i.) described a case in a man twenty years old in which a central capsular cataract was found. There was an opacity on the corresponding part of Descemet's

membrane and others at a lower level. From the upper opacity a blood-vessel ran through the anterior chamber to the upper margin of the pupil and divided about the middle of its course into five branches which entered the iris in the region of the minor circle. Beck believed that this was a congenital anomaly, while Arlt, who quoted the case in his text-book, thought that as the patient had had ophthalmia neonatorum there had been a perforating ulcer of the cornea which had left a corneal cicatrix and a deposit on the capsule. The vessel described by Beck he took to be a synechia between the capsule and the cornea.

Samelson (*Centralbl. f. prakt. Augenh.*, July, 1880) observed the following case:

In a twelve-year-old girl there was a small central macula of the left cornea from which seventeen or eighteen fibres extended to the anterior surface of the iris, which it joined at some distance from its pupillary margin. Some of these fibres divided into two or three branches. In addition there was an anterior capsular cataract which resembled a pyramidal cataract. In the right eye the macula was below the centre, and from it extended on each side, inward and outward, a thin fibril to the anterior surface of the iris. In this eye there was a posterior polar cataract which reached almost to the anterior pole; nystagmus; Hm. $\frac{1}{20}$; vision $\frac{1}{10}$. This patient had suffered a long time with ocular trouble in very early childhood, so in both cases the changes described may have been congenital, but also may have been due to perforating ulcers of the cornea caused by ophthalmia neonatorum.

Makrocki's case (*Arch. f. Augenheilk.*, xiv., p. 83) was in a fifteen-year-old girl with microphthalmos. In the right eye three fibres of the color of the iris started from the indentations on the anterior surface of the iris in the region of the minor circle and ended in the indentations nearer the periphery. On the capsule somewhat above the anterior pole was an opacity about as large as a pin's head, with a similar opacity in the cortical layer. On the cornea of the left eye, some-

what upward and inward from its centre, was an opacity composed of four separate points arranged in a crescent the concavity of which was toward the centre. These points lay in Descemet's membrane and from them extended synechiæ to the region of the minor circle in the upper inner sector of the iris. These synechiæ were of the same color as the iris and were very slender. Two of them remained single throughout, one divided into two branches, the other into three. Myopia 8 dioptres. Vision $\frac{5}{24} - \frac{5}{18}$. Posterior staphyloma in each eye. Makrocki was convinced that this was a true case of the so-called persistent pupillary membrane, because the synechiæ were equally thick, began not on the margin of the pupil, but externally to it on the anterior surface of the iris, were in the form of single fibres and were colored like the iris. He regards Samelson's case as one of faulty development in which the original symptoms may have been masked by the subsequent disease of the eyes.

In Zinn's case (*Klin. Monatsbl.*, 1890, p. 288) a leucoma could be seen by focal illumination in the right eye of a girl thirteen years old. The perfectly normal iris was bluish gray in the upper outer part, rust-colored elsewhere, with a groove on its anterior surface 1½*mm* from the pupillary margin. From the outer side of this groove sprang four fibres colored like the iris, which soon united into one fibre and passed through the anterior chamber obliquely in an inward and forward direction to the cornea. This fibre grew gradually more and more slender and assumed a rust-brown color before it reached the cornea and was lost in the outer margin of the leucoma. Somewhat below the four fibres mentioned a fifth fibre rose from the groove and to the inner side four short, rust-colored points projected into the anterior chamber. In the left eye there was a similar opacity of the cornea and a similar groove on the anterior surface of the iris. Both lenses were cloudy.

Wintersteiner (Michel's *Jahresb.* for 1893, p. 282) describes still another case in which ophthalmia neonatorum had produced a central corneal cicatrix and an anterior capsular cataract. The fibres of a persistent pupillary membrane were attached to the corneal cicatrix.

In Vossius's first case (*Beitr. z. Augenheilk.*, i., 9, 1893), a

boy twelve years old, there was a small deep opacity near the centre of the cornea which was joined to the iris by a gray, brown fibre, which arose from three points in the region of the minor circle and had two attachments to the cornea, while in the middle it was single. As there were no traces of a past iritis, or of a perforating ulcer of the cornea, Vossius considered this to be the remains of an embryonal pupillary membrane. In a second case, a male nineteen years old, he found nystagmus-like movements of the eyes, displacement of the pupils outward, and hemispherical corneæ. The sclera covered the temporal side of the right cornea for about 3*mm.* The chambers of the eye were reduced in size and the iris was adherent to the leucoma in the periphery. Near the medial margin of the leucoma was a brown stain on the posterior surface of the cornea, colored like the iris, which was atrophic. Slender fibres extended from the upper outer quadrant of the iris near the minor circle to the posterior surface of the cornea and were attached to the spot of pigment. Thick brown fibres extended directly forward from the margin of the pupil to the circumscribed opacity in Descemet's membrane. In addition to the spots already mentioned on the posterior surface of the cornea there were four punctate brown and two gray stains. No posterior synechiæ were observed. In the left eye the sclera covered the inner side of the cornea reaching nearly to its middle. The iris was adherent to the leucoma and the posterior surface of the cornea near the inner margin of the leucoma was pigmented. The iris was more atrophic than in the right eye. Vossius considered the fibres in the right eye to have been "threadlike remains of the pupillary membrane," and that the development of the eye had been obstructed so that the separation between the cornea and the pupillary membrane had been discontinued. The fact that in both eyes the sclera covered part of the cornea he explains as due to insufficient differentiation between cornea and sclera.

Wüstefeld (*Zeitsch. f. Augenheilk.*, Bd. iv., 1900) described two cases. In the first, a boy ten years old, an irregularly shaped opacity could be seen in the deep part of the left cornea. Below, bordering this opacity, was a rusty brown stripe of pigment from which extended brown threads, divided

into two groups, to the iris to be inserted in the minor circle. The iris itself was fawn brown while the threads leading to the cornea were rather darker, a rust brown. The pupil was round and reacted well to light while the threads stretched and relaxed with its movements. On the posterior surface of the cornea was also a fine curved line of pigment. The right eye was normal.

In his second case he found in the right eye of a woman thirty-six years old an opacity measuring *2.5mm* which involved the entire thickness of the lower part of the cornea. To this opacity were attached four brown fibres which arose in the region of the minor circle of the iris. Above their place of insertion in the posterior surface of the cornea were two little brown spots. Anterior polar cataract was also present. In the left eye there was a circumscribed cloudiness chiefly confined to the deep layers of the centre of the cornea and a punctate anterior polar cataract.

I have had the opportunity to observe the following case:

A. S., twenty-five years old, distant vision bad from childhood, especially with the right eye. Investigation of the right eye revealed myopia 5 dioptres; vision $\frac{4}{10}$; lids normal; two small overlapping opacities which involved Descemet's membrane alone, or at least only the innermost layers of the centre of the cornea. The rest of the cornea was perfectly transparent. On the lens capsule, somewhat above its centre, was a whitish opacity of greater extent. There were no other lenticular changes. The iris was bluish gray; on its anterior surface not far from the pupillary margin was a rather dark zigzag line. The pupillary zone within this line was covered by radiating lines placed closely together. The zigzag line was produced by little triangular, rather prominent folds in the ciliary region of the iris. In two places, below near the vertical meridian and above more outwardly, these little folds were broader, more prominent, and ended in slender threads of the color of the iris which passed through the anterior chamber and were attached the one to the upper, the other to the lower opacity on Descemet's membrane. When the pupil was dilated these threads were straight, when

it was contracted they became twisted. The fundus was normal. On the temporal side of the papilla was a small sharply defined crescent.

Left eye: myopia 4 dioptres; vision $\frac{10}{10}$; pupil crescentic with the diameter downward. The zigzag line and the division into two zones was marked only in the upper half of the iris while the lower half was uniform. It was interesting to note that in dilatation and contraction of the pupil the normal changes were observed only in its upper part, while in the lower part the radius decreased somewhat in dilatation of the pupil and increased in contraction. The probable explanation of this is absence of muscular tissue in the lower half of the iris.

Von Hippel is inclined to ascribe an inflammation or perforation of the cornea during embryonal life which caused a temporary abolition of the anterior chamber and an adhesion of the pupillary membrane to the cornea as the cause in those cases in which fibres of a persistent pupillary membrane have been observed to be inserted into the cornea. In fact it was only in Makrocki's case and in Vossius's first case that nothing could be found indicative of an inflammation during embryonal life or in early childhood. In Vossius's second case there were profound changes and in the cases reported by Beck, Samelson, and Wintersteiner there is no doubt that the changes appeared after birth. In Zinn's description it is not made clear whether the macula involved the entire thickness of the cornea or only its innermost layers. In Beck's and Samelson's cases the fibres were attached not to the margin of the iris but to its anterior surface in the region of the minor circle. But this condition can hardly solve the question whether they were anterior synechiæ or so-called persistent threads. Not very long ago I had an opportunity to observe the development of an anterior synechia in a child which had exactly the appearance of a persistent thread and was attached to the iris in the region of the minor circle.

In Vossius's case, Wüstefeld's first case, and in mine the opacities involved only Descemet's membrane, or at any rate only the innermost layers of the cornea, while the remaining layers were perfectly transparent. In Wüstefeld's second case the opacity involved the entire thickness of the cornea, but in the other eye there was an indubitable developmental fault. Furthermore the fibres joined the iris in the region of the minor circle by little pedicles and were of the same color as the iris. These conditions give reason to believe that a developmental anomaly was present in each of these four cases.

It is not difficult to explain from an embryological standpoint the origin of this anomaly. After the development of the secondary optic vesicle mesodermic cells penetrate between the ectoderm and the embryo lens which comes from it and from a common embryonal tissue from which the cornea, Descemet's membrane, and the iris, together with the pupillary membrane, are differentiated. This embryonal tissue is, I repeat, at first common to all and the division into separate layers comes later. If now the pupillary membrane, or a portion of it, does not develop normally but, as I have said above, follows the type of the iris, it may persist in the pupillary region and be attached to neither the cornea nor the anterior capsule, as many observations show. The persistent fibres are often attached to the lenticular capsule and were attached to the cornea in these four cases at least.

This attachment or, to be more exact, this union with the cornea is not very easily explained. In the case studied anatomically by Van Duyse the little plaque of lens capsule where the fibres were attached had the appearance of a pyramidal cataract surrounded by connective-tissue cells, pigment cells, and blood-vessels, one of which contained red blood corpuscles. Such formations correspond too little to the normal structure and show beyond doubt an abnormal differentiation in

the development of the eye. This abnormality resulted in the development of elements which could not atrophy in the manner of the normal elements of the pupillary membrane. The cornea and Descemet's membrane form only the outer lamella in the embryonic tissue which they have in common with the iris and pupillary membrane. They lie in opposition for a long time because the anterior chamber is developed at a comparatively later period. It would seem therefore that an adhesion of the pupillary membrane could take place more easily to Descemet's membrane than to the capsule, because in the latter case the tissue must unite with a structureless capsule, while in the former it comes in contact with the endothelial cells covering Descemet's membrane.

Krukenberg has given an analogous explanation of the origin of purely congenital melanosis corneæ, which is closely related to that of the above quoted cases.

REPORT OF MEETINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. H. W. WOOTTON, Secretary.

MONDAY, APRIL 20, 1908. DR. W. E. LAMBERT, PRESIDENT, IN THE CHAIR.

Present, 26 members.

Dr. John Leshure presented **an instrument for testing the sensitiveness of the cornea.**

The following is an abstract:

The appliance referred to consists of a piece of capillary rubber tubing similar to that found in the vaccination sets supplied by the New York Board of Health.

Its inside diameter measures about one-half millimetre, and a convenient length is two metres. The test is carried out by connecting one end of the tube to a compressed-air receiver fitted with a reducing diaphragm and cut-off, and directing a stream of air against the cornea or other structure to be investigated. It will be seen that the column of air will be very minute, and necessarily under low pressure from the friction of the walls of a tube very long in proportion to its diameter. By varying the regulating diaphragm the pressure can be reduced or increased and the test thus be made quantitative. A column of air emerging from the distal end of a tube two metres long, under a pressure of five pounds in the receiver, should be distinctly, though slightly, felt by the normal cornea. Any cornea not perceiving such an impulse should be considered anæsthetic.

This test should be of value in cases of dementia præcox where anæsthesia of the cornea is an early symptom, and

is rather more delicate than any test which necessitates touching the cornea with a hard body. Areas of anæsthesia in the region of the temple, lids, conjunctiva, etc., can also be mapped out accurately.

By immersing the tube in hot or cold water, the air can be warmed or chilled, and the senses of heat and cold tested. An almost equally satisfactory way of making the test is by blowing through the tube, first connecting the latter with a glass mouthpiece in which is placed a little cotton to intercept moisture or foreign matter. By using tubes of varying length the pressure can be increased or reduced, as the latter is inversely proportional to the length of the tube, and a quantitative element introduced in this way.

Dr. E. B. COBURN presented **an apparatus to measure the ocular tension.**

The apparatus consists of three parts—an applantation instrument, a rubber ball, and a mercury manometer.

The applantation instrument consists of a glass tube, about 6*mm* in diameter, drawn down to a capillary tube in the centre. This is surrounded by a brass sleeve, fenestrated in the middle part so that the capillary tube may be seen, and provided with a sliding marker which points toward the capillary tube. One end of the glass tube is covered with a thin rubber membrane, while the other end is connected by rubber tubing and a three-way tube to the rubber ball and the manometer, the ball being connected between the instrument and the manometer. The glass tube is filled with a colored fluid to the lower end of the capillary tube and the marker is adjusted to indicate the top of the fluid.

The instrument is now applied to the eye with just sufficient force to barely cause an indentation of the ocular tissues. This causes the rubber membrane to be pushed inward, causing the fluid to rise in the capillary tube. Pressure is now made on the rubber ball until the fluid returns to its original level as indicated by the marker, and the manometric reading is taken. This will indicate the ocular tension. Corrections may be made for the weight of the water indicator and for the elasticity of the rubber diaphragm, but these values are small and may be neglected.

Dr. H. H. TYSON presented a case of **congenital fistula** of

the lacrymal sac. Published in these ARCHIVES (vol. xxxvii., No. 4, pp. 396–401).

Dr. S. PAYNE presented a **patient upon whom he had operated for restoration of the lower lid.**

Patient was admitted to the Manhattan Eye, Ear, and Throat Hospital February 28, 1908. Twenty-two months previously he had been injured by a premature explosion of dynamite. As a result, the lower jaw was fractured, also his left tibia and fibula and right radius and ulna. His right eye was completely enucleated and eyelids torn off. On a more careful examination, a very small part of the eyeball was found remaining, with adhesion extending from this upwards to the upper lid. The middle third of the upper lid was torn away, and the outer and inner thirds drawn upwards by three scars, each extending far above the eyebrow. The lower lid was entirely torn away, the raw surface extending half an inch or more below the orbital edge. The day he was admitted to the hospital the adhesion already described was cut, in order to see what effect it would have on the upper lid.

March 4th.—Under ether, a complete undermining of scar tissue to an inch or more above the brow, and the two sides of the torn lid were brought together with sutures.

March 11th.—Remaining portion of the eye was enucleated, with some difficulty on account of extensive adhesions. The operation on the lower lid was reserved to the last, in order to study the best way to get the best result. The punctum remained at the lower edge of the raw surface, and the conjunctiva was drawn tightly across the edge of the orbit, and the entire raw surface was covered with conjunctiva.

March 25th.—Under ether, beginning on the nasal side, the punctum was dissected out with scissors, so as to leave it lying towards the conjunctiva. The incision was then made the entire length of the lid, cutting down for some distance also in the nasal and temporal direction. The conjunctiva was then dissected upwards until it had dropped back into the orbit, and was then taken up by three stitches and fastened to the upper lid. A measurement was roughly taken of this surface preparatory to removing the skin from the arm. The skin removed was placed in position. A curved needle, with thread, was passed through the skin of the face, not

entering the skin-graft but passing over it and into the edge of the conjunctiva, and tied across or over the skin-graft. Three stitches were used, one at the centre and one at each end. The graft was wide enough to allow for contraction. The result is that he has a lid that will hold a glass eye, with the punctum about as nearly in its normal position as is possible.

As the operation on the upper lid was a failure, Dr. Payne thought his next attempt would be to replace this lid also with skin-graft from the arm.

Dr. W. E. LAMBERT presented a patient upon whom he had operated for **epithelioma of the inner canthus.**

The tumor was removed and the patient was afterwards treated by Dr. Dixon by means of the X-ray. On June 17, 1907, she presented herself for X-ray treatment. She received six exposures from a medium low tube from six to ten minutes each, at a distance of nine inches, strength of current 9 amp. The wound was entirely closed June 26th, and patient was discharged July 1, 1907.

Dr. ARNOLD KNAPP presented **an apparatus to determine the enlargement of the blind spot in Bjerrum's method of testing the field of vision.**

Dr. ARNOLD KNAPP presented a case of **melano-sarcoma of the conjunctiva,** which he had removed from a woman sixty years of age. The tumor started apparently in a pigmented spot near the caruncle, which slowly enlarged, forming a small lump under the lid. During the last four months it has grown rapidly, and before operation occupied the region of the entire upper lid, involving the palpebral conjunctiva and the tarsus but not the overlying skin; there was also a flat extension in the palpebral conjunctiva of the lower lid.

Dr. E. L. OATMAN described **a method of rapid fixation and hardening of eyes for microscopic examination.**

The present popular method of preparing eyes for microscopic examination with formalin and alcohol is very satisfactory. It is, however, subject to certain limitations: (1) it is very slow and expensive; (2) alcohol cannot be used as a fixative because, being hygroscopic, it will rapidly extract water from the vitreous and cause great shrinking of the globe;

(3) some valuable fixatives with low penetrating power cannot successfully be employed without opening the eye and destroying the relation of parts. The simple device here described seems to overcome the difficulties mentioned. The principle employed is to supply alcohol or other fluid to the interior of the eye as rapidly as the intraocular contents are abstracted by the osmotic action of the bath in which the eyeball is immersed. This is accomplished by means of a fountain syringe containing the fixative, which is connected with the interior of the eye by a hollow silver needle and rubber tube. I do not know that this is a better, or even as good a method for general use as the one at present employed, but it will be found useful where speed and certain special methods of fixation are required. I am not as yet prepared to state how the best results are to be obtained with it. I have done well with the following procedures: Formalin 10% for a few hours, followed by alcohol 95%; Zenker's fluid followed by washing and alcohol; alcohol 95% in the fountain and 80% in the bath, followed by alcohol 95% in both. Eyes may be bisected forty-eight hours after commencing the alcohol.

In the discussion of Dr. Coburn's apparatus, Dr. A. KNAPP asked how the pressure employed in holding the cylinder against the eye could be regulated. Dr. COBURN replied that it seemed only necessary to hold the cylinder so that the ocular tissues just began to dimple. Dr. W. E. LAMBERT asked whether the difference of pressure exerted upon the bulb would not cause variation in the readings. Dr. COBURN explained that the degree of pressure could not always be the same but must vary, according as the eye was soft or hard.

In the discussion of Dr. Knapp's specimen, Dr. E. L. OATMAN stated that he had seen a similar case operated upon without return.

In reference to Dr. Tyson's patient (congenital fistula of the lacrymal sac) Dr. J. E. WEEKS had seen two cases. In one, the fistula was unilateral—in a girl eight years of age. The orifice was so very minute that it was almost impossible to see it. The second case was also unilateral. These cases were always caused by developmental failure in the closure

of the grooves from which the lachrymal passages were formed.

There was no discussion of Dr. Payne's patient.

In regard to Dr. Lambert's case, Dr. J. E. WEEKS had seen a case of epithelioma of the inner canthus many years ago, which was operated upon by a general surgeon. The tumor was simply removed by two curved incisions, leaving a fresh surface two-thirds of an inch in diameter. A simple gauze dressing was applied. It healed up perfectly, with as good a result as in Dr. Lambert's case, although no X-ray had been employed. Dr. ALEXANDER DUANE asked if, in general surgical practice, the X-ray was still employed after removal of carcinoma of the breast. Dr. GEORGE S. DIXON stated that the idea of using X-ray was to hasten filling in of the defect, and this he believed it did with less shrinking than when it was not employed. In reply to Dr. Duane, he believed that the X-ray was still used in general surgery after removal of carcinoma.

In the discussion of Dr. Arnold Knapp's apparatus, Dr. ALEXANDER DUANE stated that he was personally partial to a large screen which could be used for other purposes, *e.g.* scotomata, and the determination of the field of fixation. The screen need not be more than five feet square. For a small screen, Priestley Smith's scotometer was probably the best and most simple.

In regard to Dr. Oatman's method, Dr. GEORGE S. DIXON thought the principle excellent for rapid work, and it would be most useful for fixing the retina if we were in a hurry to make the examination.

Dr. J. E. WEEKS reported the method that he employed in plastic operations on and about the eye by the use of flaps of skin (Wolfe flaps) and mucous membrane without a pedicle, and Thiersch grafts. He also demonstrated on the cadaver the operation for the reconstruction of the margin of the lid by the implantation of a strip of tissue into an inter-marginal groove; also the operation for the restoration of the conjunctival cul-de-sac in cases of partial or total loss of conjunctiva. Dr. Weeks reported eighteen successive operations of the latter variety, all of which were successful. Photographs of patients illustrative of results were presented.

In the discussion of Dr. Weeks's paper, Dr. A. KNAPP had been much surprised at the great amount of shrinkage that took place in one of his cases, so much as to cause entropion, although the flap had been as large as could possibly be used. He had had a good deal of trouble in getting the lateral margins, particularly that towards the nose, to heal. Dr. E. GRUENING stated that Dr. Weeks's operation was far superior to the transplantation of Thiersch grafts. It was easier to trim the flap, that is to say, to remove the subcutaneous tissue at the same time that the flap was cut and before it had been entirely detached from the patient. He even put the sutures through the flap before detaching. He thought that Dr. Weeks could certainly claim priority in the use of Wolfe grafts for the restoration of the cul-de-sac. Dr. W. E. LAMBERT had seen ptosis following one case. Dr. WEEKS had had no difficulty with entropion following the operation. Shrinking at the inner and outer margins could be obviated if the sutures were passed through the canthal ligaments. The depth of the sac was maintained in every case. Dr. E. Gruening's method of trimming the flap *in situ* was a good one. The ptosis was inevitable if the flap was attached to the orbital margin above, for the levator must be cut, but this did not interfere with the appearance produced while using an artificial eye.

MONDAY, MAY 18, 1908. DR. W. E. LAMBERT, PRESIDENT, IN THE CHAIR.

Present, 48 members.

Dr. J. E. GILES presented a case of **insufficiency of convergence with unusual features.**

Dr. Giles first saw the patient in July, 1906. She complained that she was always tired after using the eyes, and that glasses had never given her any relief.

O.D. $\frac{20}{100}$ and $\frac{20}{30}$ with $-.50-2.25$ c. ax. 10.

O.S. $\frac{20}{40}$ and $\frac{20}{30}$ with -2.25 c. ax. 180.

Patient presbyopic and read Jager No. 1 at ten inches, with + 2 sph. added to the above correction. Exophoria four degrees.

October 31, 1906.—Reported that she could not wear the

glasses comfortably, and is uncomfortable either with or without the glasses. Dr. Giles prescribed prisms, two degrees each, to be worn base in before her glasses.

October 20, 1906.—Showed total exophoria of seven degrees, but had had some relief from the prisms. Inquiry revealed that both externi had been divided twelve years ago in San Francisco. In spite of this she had insufficiency of convergence, with diplopia at the near point.

About November, 1906, she visited an institution in this city and the surgeon who examined her performed a tenotomy of the right externus.

Nov. 25, 1907.—Patient showed esophoria four degrees with the phorometer and six degrees with the Maddox rod. Abduction one degree, adduction nineteen degrees. No hyperphoria. Sursumduction, right, two degrees; left, two degrees. Has homonymous diplopia at twenty feet if she looks to the right of the median line, but has crossed diplopia at any point nearer than twelve inches (sometimes even at twenty-four inches).

Patient states that she has been advised to have the other externus divided, but had been afraid to have it done.

In consultation with Dr. W. R. Broughton, it was decided to advance the right externus. This was done early in December, 1907, and at the last examination the patient showed abduction eight degrees, adduction twenty-four degrees, exophoria one-half degree. Dr. Giles stated that the most interesting point in this case was the increase in adduction after the advancement of the externus, and he also stated that the case illustrated very well the necessity of a minute examination of the action of all the eye muscles before any operation was performed. In regard to the increase of converging power after the advancement of the externus, Dr. Giles thought that it was to be explained by the fact that the previous tenotomy of this muscle had caused a great number of adhesions, which prevented satisfactory convergence. These, of course, were divided at the same time the muscle was advanced, and thus a cure, not only of the esophoria due to a previous tenotomy, but also of the exophoria, due to the convergence insufficiency, resulted.

Dr. W. R. BROUGHTON believed that Dr. Giles's explanation

of the remarkable increase in adduction after the advancement of one externus was entirely correct, *i.e.* that the limitation in the rotation both in and out was due to the adhesion of the muscle. When he operated he found not only ordinary adhesions, but a complete attachment of the whole posterior surface of the external rectus of the globe, so that he had to dissect it from the ball back beyond the equator. This would limit the rotation both inward and outward. The only wonder was that after the muscle was reattached it did not again adhere to the ball and neutralize to a large extent the whole result. Whereas the patient could not look one degree to the right without homonymous diplopia or converge nearer than twelve inches without crossed diplopia, she now had binocular single vision in every field except at the extreme right, where she had a slight homonymous diplopia. The adduction now was 40 and the abduction 8, and she can converge up to eight inches. She is using her eyes all day as a seamstress.

Dr. H. W. WOOTTON thought that the case also illustrated very well the inadvisability of performing tenotomies of the externi for insufficiency of convergence. In his opinion, the proper operation in the beginning would have been in advancement of one, or possibly both interni. Tenotomy of the externus would not cure insufficiency of convergence. As a matter of fact, it usually added to the patient's difficulties by producing esophoria for distance due to a divergence insufficiency paretic in character and depending upon the tenotomy. This is exactly what had happened in Dr. Giles's case. After the tenotomies inability to converge still persisted, and the patient was made uncomfortable by the homonymous diplopia for distance, which had resulted from the tenotomy.

Dr. H. H. TYSON presented a case of **horizontal oscillations of the globes in attempted closure of the lids, in facial palsy.**

J. K., age fifteen years, had an attack of right facial palsy nine months ago. Present condition: Vision, right eye, varies from $\frac{20}{40}$ to $\frac{20}{25}$ due to spasm of accommodation; left eye, $\frac{20}{20}$. Motility, each eye tested separately, normal. Conjugate movement to left exceeded same movement to right by fifteen degrees. Orthophoria distance—Exophoria one-

half degree near. Fusion power muscles for distance—adduction, twelve degrees; abduction, four degrees; sursumduction, three degrees; deorsumduction, three degrees. Candle test shows no diplopia. Upon forced movements of eyes to extreme right or left, pseudo-nystagmus was produced, sometimes horizontal, at other times mixed. Upon attempting to forcibly close right eyelids, his eyes would move up and to the left, and horizontal oscillatory movements ensued with the eyes moving through an arc of about thirty-five degrees, extending from a point five degrees to the right of the median line to a point about thirty degrees to the left. This movement seems to be due to an overflow stimulus from the facial nuclei to the third nuclei, through the longitudinal fasciculi, and also to the centres for associated movements. The conjugate movement to the left in this case being the stronger, we thus obtain our nystagmic movement in that direction, which might be termed an associate nystagmus. H. Schlesinger, who reported a somewhat similar case, stated that in all probability the horizontal movements of the eyeballs were due to central disturbances of innervation. An analysis of this case would indicate that it was due to an overflow impulse as heretofore stated, and to an insufficiency in the conjugate movement to the right. Stransky reported a case of rapid oscillation of the eyeball, either in a horizontal or oblique direction, synchronous with contractions of the orbicularis when resistance was offered to its contraction with the fingers, occurring in a neurotic individual. The pupils in this case were at times equal, at other times unequal, the right being the smaller, due to spasm of the sphincter pupillæ. Light and convergence reflexes normal. Corneal sensibility diminished in right eye. Color fields gave reversal of fields, red and green being largest and white the smallest, indicating a hysterical element as a complication.

Dr. H. H. Tyson also presented a case of **cyanosis retinæ** occurring in general cyanosis due to a congenital heart lesion, which was diagnosed as patent foramen ovale with pulmonary stenosis. A full and detailed report of the case appears in this number, page 555.

There was no discussion of Dr. Tyson's cases.

Dr. Ervin Török read a paper entitled **The diagnostic**

and therapeutic value of tuberculin in the treatment of eye diseases. Published in full in this number, page 531.

In the discussion which followed, Dr. N. B. POTTER outlined his opinion of the value of the opsonic index in controlling the use of tuberculin. The following is an abstract:

It is well known that many objections have been emphasized in America. The technic is so difficult and time-consuming that but few laboratory men will acquire the necessary facility and accuracy. Even a skilled worker can hardly expect to estimate more than eight or ten indices per day. This makes each estimation expensive. Another obstacle is that the clinician must needs wait until the next day for the figures. So when the patient presents himself for inoculation his opsonic index cannot be at once utilized to guide the advisability or the size of each dose. Further, the opsonic index represents an ingenious method of estimating the phagocytic activity, but it measures neither the agglutinating, bactericidal power, nor many other properties of the blood which have to do with the production of an active immunity. Finally, our experience goes to prove that it is not possible to obtain any high degree of accuracy with this method. Last year Dr. North sent ten duplicate series of twelve tubes of blood to each of ten laboratories for determination of the opsonic index. The series consisted of three specimens from one individual, four from a second, two from a third, one from a fourth, one from a fifth, and two specimens of heated serum from the fifth individual. None of the laboratories furnished consistent opsonic indices and none of them agreed. Yet the laboratory workers who performed these tests had been engaged upon this technic for from six to eighteen months. On the other hand, despite these rather grotesque results, there is usually a very striking uniformity in the opsonic determinations when made by an individual worker well trained in the technic, and the indices seem to correspond rather closely to the clinical progress of the patient. The following figures will illustrate its relative accuracy:

Collier, at St. Mary's Hospital, sent to ten laboratories eleven specimens of blood taken at the same time from a case of tuberculous cystitis. The tuberculo-opsonic indices follow: .84, .87, .79, .87, .94, 1.04, 1.07, 1.09. 1.10, 1.13, 1.7. The first

four indices were made by men trained in the same laboratory; the last by a worker in a laboratory where the opsonic theory is discredited.

RELIABILITY OF THE TUBERCULO-OPSONIC INDEX.

NORMAL INDIVIDUALS.

OBSERVER.	NUMBER OF DETERMINATIONS.	BETWEEN 0.8 and 1.2
Park,	18	72 %
Fried,	52	87
Reyn & Kjer-Petersen,	673	72
Wright's Laboratory,	635	100

TUBERCULOUS PATIENTS.

Urwick (pulmonary cases)	32	
Fried (mixed cases)	74	
Reyn & Kjer-Petersen,	408 (between 0.90 & 1.16 43%)	about 59 %
(lupus)	(0.77 & 1.29 73%)	
Western (lupus)	80 (between 0.85 & 1.15 40%)	

Last year we attempted to assist Doctors Cutler and Duane in the administration of tuberculin to three cases of probable ocular tuberculosis by estimating the opsonic indices. We were unable to satisfy ourselves that these determinations were of any particular value in guiding the dosage or the interval of inoculation. Last year in a paper read before the Association of American Physicians at Washington, I ventured upon the following statement in my conclusions:

1. Wright's method of estimating the opsonic indices in bacterial infections is hardly accurate enough to compensate for the amount of time involved in its application. There is a general parallelism, however, between the opsonic indices and the clinical course of most cases, so that these determinations should be made when the results of inoculation cannot be controlled by the ordinary clinical methods of observation, when there are several organisms present, and when the patient does not improve under the usual method of inoculation.

Further experience and a careful review of the literature upon the subject have induced me to abandon almost entirely opsonic determinations; and in this decision I am in accord with the majority of the more careful American observers experienced in the technic.

Dr. WILLIAM H. PARK stated as follows:

It is my purpose to confine my remarks upon the paper of Dr. Tŏrŏk as far as possible to the bacteriological side. With Dr. Potter's statement upon the value of the opsonic index I fully agree. We have been associated in this work for the past year, and have come to a common understanding upon many of the points involved.

In a visit to Wright's laboratory last summer, in which he very kindly showed us the results of using the opsonic index in the guidance of therapeutic inoculations, we found it impossible to satisfy ourselves that the index really gave any help, nor could we see that when the inoculations were given upon an index very different from what it had been supposed, any harm resulted. At Saranac Lake numerous tests have been made and no regular changes in the opsonic index have been seen to follow doses of tuberculin, as small as are now generally agreed upon as being suitable for treatment. I think also that it is safe to say that the majority of observers, who have not come directly under the influence of Wright, do not think that variations in the opsonic index are sufficient to make a diagnosis in very slightly developed cases—that is, in those cases in which the diagnosis could not be made by the usual means.

As to the therapeutic value of tuberculin, my own experience has not been in tuberculous eye diseases, but the general principles certainly apply to all forms of tuberculosis. I think we are safe in asserting that the best observers have become firmly convinced that the very small doses of tuberculin, such as have been advised by Dr. Tŏrŏk, do no harm, and in the majority of cases certainly do good.

The more localized the tuberculosis and the slighter its amount the better are the results. As you know, for diagnostic purposes we confine ourselves to the filtrate of the boiled broth culture of tubercle bacilli. This fluid, which is the original tuberculin, contains the substances which give the diagnostic reaction, but through boiling has lost certain substances which are believed to be valuable in immunization.

For diagnostic purposes, we have the subcutaneous injection, the cutaneous inoculation, and the eye inoculation. These three give very similar information as to the presence of tuberculosis. All three of them give a reaction in persons

who have a recently healed tuberculosis, as well as those having active tuberculosis, as the reaction is one of sensitizing and not of poisoning.

The subcutaneous injection gives not only a general reaction but usually a local reaction about the tissue infected. A general reaction, of course, only indicates present or recently healed tubercle deposits in some part of the body, while a local reaction at a distance from a subcutaneous inoculation indicates the place of infection. For diagnostic purposes in suspected eye-tuberculosis, this local reaction would probably be especially valuable. The application of the tuberculin to the conjunctival mucous membrane, or to the skin, gives very similar results, both indicating when the reaction occurs merely the presence somewhere in the body of a tubercle deposit. A reaction from the less concentrated solutions of tuberculin is believed to indicate a more active condition than from the more concentrated solutions; thus, one from a cutaneous inoculation with a ten per cent. solution of tuberculin suggests an active deposit, while a negative reaction with a ten per cent. solution and a positive reaction with a twenty per cent. solution would suggest a healed tuberculosis.

The experience gained by many thousands of cases inoculated indicates that a definite reaction only occurs in those who have recently had or have at the time of inoculation a tubercle deposit.

Cases with moderate localized tuberculosis generally react more than those with advanced tuberculosis, and very anæmic or sick persons fail to react at all. The dangers of the eye inoculations are slight, but still present; probably about one in five hundred cases gives more or less serious after-effects. At present it is considered unwise to inoculate the eyelid if either eye is diseased, and certainly not to inoculate a diseased eye. No dangerous after-effects have followed the cutaneous inoculation. The various tuberculin preparations are in need of standarization.

Dr. A. WADSWORTH made the following statement:

The excuse for, and the essential idea in, this revival of therapeutic measures which past experience long ago discarded, are based on the supposition that in some cases the infectious agents lie dormant or are altered physiologically and so fail

to stimulate sufficient protective reaction in the body tissues for a complete immunity. By the introduction of vaccines, this deficiency is made up, and if not exceeded, the immunity of the patient is gradually and materially strengthened. Precision in this method of treatment obviously depends upon that with which the degree of immunity existing in any patient from day to day can be formulated. Accepting the phagocytic activity of the blood serum as a reliable criterion of the immunity of the patient, Wright has most ingeniously devised methods seemingly simple and practical for the determination of the opsonic or phagocytic activity of the serum.

The blood serum of the patient and that of a healthy person are each mixed with the washed leucocytes and bacterial suspensions in equal portions. After a definite incubation, these mixtures are spread on slides and the average estimated by counting the bacteria found inside the leucocytes. The average for the normal person and the patient are compared and the opsonic index is obtained by dividing the average for the patient by that of the normal person. Ingenious and valuable as these methods are in the special study of phagocytosis, other observers adopting Wright's technic have encountered difficulties insurmountable in their application to the practical routine of clinical work. The phagocytic activity, even if accurately determined, is but part of the protective mechanism, and in the different infections, or even in different individual cases, varies widely in the rôle it plays in recovery.

The difficulty in being sure of the normal for comparison also renders the significance of the results uncertain. Of late this has been in a measure obviated by using for the normal serum a pooled serum or mixture of sera from a number of healthy persons, but this also complicates, to some extent, the technic, for these normal sera it is now known should be of precisely the same age and condition as that of the patient. In the measuring of the quantities in the fine capillary pipettes, in their exposure at the proper temperatures, in the spreading of the material on the slides, and in other details, but chiefly in the counting of the bacteria in the leucocytes, there are numerous pitfalls known only to those experienced in the work, which render accurate determination uncertain. The error under the best of conditions in fact often greatly exceeds

the degree of change in phagocytic activity for the estimation of which the method is employed.

A year and a half ago in discussing Dr. Park's paper before a meeting of the Roosevelt Hospital Alumni, I called attention to the agglutination of the organisms about the leucocytes as a marked and at that time unnoted source of error. The error at that time in staphylococcus determinations with sera from animals under experimental conditions was more than 200 per cent. A year ago, in Washington, the value of these opsonic determinations was more exhaustively considered. Baldwin with the tubercle bacilli even in his routine determinations of patients' sera found the agglutination a disturbing factor. North estimated and reported the errors in the results obtained by different laboratories in their effort to determine the phagocytic activity of different sera unknown to them. No two workers reported alike on the same serum, and no one worker reported on the same serum twice alike, the minimum of error being about 25%, the maximum 200 to 500%; and the minimum of error was largely confined to workers skeptical of the value of the results.

Some attempt has been made within the year to improve the technic, and some suggestions, especially those fixing the conditions of observation, the suspensions of bacteria, the number and manner of counting the leucocytes and their bacterial contents, have been of considerable value, but certain it is that this ingenious technic of Wright, for opsonic determinations, is as yet of little or no practical value, to the clinician in the routine study of his cases. The clinician thus still finds his bedside observation the only guide to the quantities of vaccine to be injected and the interval between the doses. But this injection of bacterial cells at intervals it is now known is not altogether free from danger. The inoculation of an animal with the albumin or proteids from other animals or bacteria, if made daily, gives rise to a specific immunity, but if spaced at a longer interval than two weeks or possibly ten days, the second inoculation may give rise immediately to grave symptoms and death; a hypersensibility develops under these conditions and this hypersensibility may persist a long time. All this knowledge of anaphylaxis opens up a wide and an extremely important field of investigation, and casts upon this

promising revival of specific vaccine therapy and diagnosis, at the very inception of its reorganization, a doubt as to just how the best results are to be secured with it and the dangerous ones avoided. A syringe with seemingly benign or curative sera and vaccines in the hands of the inexperienced is to-day, as in the past, a dangerous thing, and unfortunately the inexperienced and more credulous step boldly forth into this field where their more experienced colleagues have feared to tread. But the proof of the value of measures such as the conservative administration of tuberculin in tuberculosis, especially in the more definitely localized forms, lies in the beneficial, even curative, results that have already been recorded, and the infrequent occurrence of very serious or harmful mishaps. These mishaps, as our experience with tuberculin therapy in minute doses increases, will doubtless diminish in number and possibly may be rationally safeguarded by advances in our knowledge of the subtle conditions underlying the phenomena of anaphylaxis.

Dr. C. S. BULL advocated conservatism and the thorough consideration of the subject before becoming too enthusiastic. About a year ago he had reported ten cases in which tuberculin, both the old and the new, had been used for diagnostic and therapeutic purposes. His deductions had been about the same as Dr. Török's. Since that time he had had seven additional cases which had confirmed his previous views. From what experience he had had the ocular reaction seemed to be of considerable value, but also of considerable danger. He had seen violent reaction in two cases. The ocular test produced no general disturbance. The subcutaneous diagnostic method, when followed by both a local and general reaction, meant tuberculosis, except that it had been stated that in very young children no reaction might follow, although the patient might be tuberculous. He had used injections of *1mg* for diagnosis and had given as high as *5mg* during treatment. If two injections were given for diagnostic purposes, the local reaction in the eye might be expected after the second. The value of the therapeutic injection was not easy to understand, for the immunity developed was relative and limited. The bacilli in the eye are not killed. The tuberculin merely causes the production of scar tissue.

Relapses are frequent, and treatment should be long-continued. He had seen ciliary injections persist for some months after disappearance of all other symptoms. Von Hippel's statement, that all cases, no matter how serious, were curable by the use of tuberculin, was too optimistic. He had had two cases in which the treatment absolutely failed.

Dr. P. A. CALLAN stated that about five years ago he had used tuberculin by subcutaneous injection for diagnosis. He had seen reaction in undoubted cases of syphilitic eye disease. As a diagnostic method it was uncertain, and harm could be caused by it. He had seen a sub-normal temperature of even two degrees follow a couple of hours after the injection. He had also seen a reaction occur as late as the fifth day. For himself, he saw very few cases of tuberculous disease of the eye, and was surprised to find them so numerous in the practice of others. He thought many of the cases supposed to be tuberculous were really syphilitic cases. As a therapeutic measure, it gave good results.

Dr. L. E. HOLT made a report upon 586 eye tests with tuberculin carried on at the Babies' Hospital. A one per cent. solution was used in nearly all cases. In forty-two cases positive results were obtained. One of these was shown by autopsy not to have tuberculosis, and two others probably did not have it. Doubtful reactions occurred in ten cases. Most of these, however, were very slight. Three were positively tuberculous, and one more probably so. Negative reactions were obtained eight times in tuberculous patients, all of these being either dying children or those suffering from extreme prostration. In the main, therefore, the test gave reliable results. In the forty-two positive reactions, symptoms were noted in less than twelve hours in all. The usual time of beginning was from six to eight hours. The reaction reached its height in twenty-four hours in thirty-eight of forty cases. The duration was less than twenty-four hours in twelve cases; two or three days in twenty-one cases; four to six days in three; seven to ten days in two. Of the children reacting positively, fourteen were under one year old; six of these being under six months, the youngest, two months. This disproves the statement often made that young infants do not react. No injurious effects were noted in any case.

The most characteristic thing about the reaction was the abundance of secretion present and the regular course of the symptoms.

Dr. J. E. WEEKS stated, in regard to the diagnosis of tuberculosis in the eye, that the method by removal of tissue or of secretions and examination of these was very difficult in tuberculosis of the cornea, iris, or sclera. It was only occasionally that sufficient tissue could be obtained for a satisfactory examination. He had at present under his treatment a case of supposed tuberculosis of the cornea. The removal of pus from a small abscess in this case showed no tubercle bacilli. The method of diagnosis by exclusion, that is to say, the diagnosis of tuberculosis after syphilitic treatment had failed, warranted the institution of treatment by tuberculin. In fact, he thought it should always be tried in such cases. Certainly, if we obtained a reaction in cases in which mercury and iodide of potash had not succeeded, the case was most probably one of tuberculosis. He had treated seven cases in all, and improvement had resulted in each case. In one case of conglomerate tubercle of the iris cure was brought about by from sixty to eighty injections, extending over a period of eight months. A dense cicatrix was produced at the point where the tubercle had been present, and the iris was left bound down to the lens capsule. After injection for diagnostic purposes, a sub-normal temperature at first was by no means uncommon, and the subsequent rise might last for thirty-six hours. Therapeutically, tuberculin was of great value, and the classification of certain eye diseases had been made more perfect by its use.

Dr. ARNOLD KNAPP thought it was important to try the subcutaneous tuberculin test in every chronic form of eye disease where the diagnosis was obscure. The local reaction in the eye was of equal, if not of greater importance than the rise in temperature. The treatment with tuberculin is necessarily prolonged and often tedious. It should be more generally known that the tuberculin treatment, if properly administered, is entirely free from danger. When the eye lesion has cleared up as much as it can and the process in the eye can be regarded as cured, the question naturally arises, are the other tuberculous foci in the body also healed? On this point the aid of the internal diagnostician must be called upon.

REPORT OF THE MEETINGS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.

By Mr. C. DEVEREUX MARSHALL.

The ordinary meeting of the Society was held at the Medical Society's Rooms on Thursday, May 7, 1908, Mr. Marcus Gunn, F.R.C.S., President, in the chair.

Mr. E. E. Henderson showed microscopical sections from the case of **orbital growth** shown at the last meeting. Dr. Edridge Green exhibited and demonstrated **a lantern,** of an economical design, **for testing color perception.** Mr. Devereux Marshall highly approved of the lantern, which he understood would cost only 25 shillings, as against about six guineas for the more elaborate lantern of Dr. Edridge Green. Mr. Nettleship said he had used Priestley Smith's lamp, and found it very useful, patients making very few mistakes. The President said he had had a lamp in use for 15 years, with ordinary red and green glasses, and an opaque ground glass, with a diaphragm. Dr. Clement Hailes showed a portable illuminative attachment for the ophthalmoscope, which could also be used as a focal light. It was attached to a small accumulator which could be carried in the pocket, leaving the hands free. Mr. J. H. Tomlinson showed a Nernst-lamp projecting lantern for the consulting room, also a small Nernst lamp in which the resistance was in the handle. Mr. Jessop showed a case of **aniridia of both eyes.** Mr. George Mackay, Mr. Treacher Collins, and Mr. Devereux Marshall described similar cases. Mr. Jessop showed a boy æt. 9½ with glioma. Mr. Freeland Fergus read a paper entitled **ptosis operations.**

The author first referred to the operations by sutures

and ligatures, as illustrated by the operations of Mules, Harman, and Worth. Attempts had also been made at advancement, as in his own method of advancement of the occipito-frontalis, and to attach a portion of the superior rectus muscle to the upper eyelid. Others had tried to advance the levator palpebræ superioris. Finally, there was the plastic operation as in the well-known operation of Panas. The suture operations seemed in their essence bad: a thread which was absolutely sterile would not cause cicatricial contraction. Moreover, threads left projecting on the skin surface were apt to be channels of septic invasion. Ligature operations left the lid without much mobility, and success depended on the permanency of the metallic or tendinous suture. Of the advancement operations he was satisfied with that of the occipito-frontalis. Recently, however, he had used a much larger incision than formerly. He now made an incision right along the eyebrow, and then continued it for 1½ to 2 inches upwards and outwards, so that he could reflect the skin very thoroughly. Where asepsis was secured, the patient was not disfigured with a scar in after-life. In many cases of ptosis the levator palpebræ superioris did not seem to be developed at all. The eyeball was protected either by a Knapp's lid clamp or by an ordinary metal spatula. A skin incision was made parallel with the edge of the eyelid and across its entire extent. It was situated a few *mm* above the free border of the lid. Another incision began at one end of the primary incision, curved upwards over the eyelid, and downwards, so as to terminate at the other end of the incision. The portion of skin thus isolated was then dissected up, and the tissue down to the conjunctiva removed. One set of sutures was placed deeply, consisting of one point in the middle, and one at either side. Superficial skin sutures were also inserted. For the deep sutures he used van Hoorn's .00 gut. That operation lifted the eyelid easily to the required amount, and put it completely under the control of the occipito-frontalis muscle. Dr. Fergus showed a number of photographs of cases so treated, with excellent results.

Mr. PRIESTLEY SMITH thought the question of most importance was what happened during sleep in cases which had been operated on—there might be considerable gaping of

the lids. Mr. ROLL thought quite as good results were obtained from Hess's operation as Mr. Fergus got by his method. Mr. MARSHALL thought that during sleep the eyeball rolled upwards, and that therefore the cornea would then be covered in any case. Mr. FERGUS replied: He said he did the first operation 12 years ago, and he had not yet seen corneal trouble. He believed that closure of the eyelid during sleep was involuntary. If he should encounter a case of ulceration of the cornea following the operation he would consider it his duty to report it to the Society.

Messrs. E. CLARKE and S. MAYOU read a paper on **tubercle of the iris treated by tuberculin.** In the case there was a large area of detached retina, forming a whitish prominence on the nasal side of the disk, 3 D. at the highest point, and crossing below the large inferior retinal vein. The retina in the upper part was covered with an exudate. There was no history of tubercle or syphilis in the case. The vision was $\frac{5}{15}$, the vitreous full of opacities, and there was a mass of k. p. spots in the lower part of Descemet's membrane. A tumor was very distinctly seen, and as a result of a consultation with one or two colleagues, it was decided to ask Sir Almroth Wright to treat the case by tuberculin. That was done, and six months after commencing the treatment, improvement set in: vision increased, and the k. p. spots disappeared, and the growth became flatter. The treatment was continued for a year, during which 21 inoculations were given, and the result was to drive the tubercle out of the system. The eye was now practically normal. Mr. CLARKE next read a paper on **confluent or conglomerate tubercle of the iris treated by tuberculin.** Except for hyd. cum creta every night for a week on admission and cod liver oil, no other treatment was given. After needling, which he proposed to do later, the case was a complete success. The cases were discussed by the President, Mr. Jessop, Mr. Herbert Fisher, Mr. Sydney Stephenson, Mr. Bishop Harman, Mr. Fergus, Mr. Johnson Taylor, and Mr. Cruise. Mr. CLARKE, in his reply, said the mercury was given as he wanted to have a second string to his bow and avoid wasting precious time. He did not use Calmette's reaction, as he knew of a case in which inflammation of the eye had persisted for some weeks after its use.

Dr. GEORGE MACKAY (Edinburgh) read a paper entitled **Note on a case of arterio-venous aneurism treated by ligature of the common carotid artery.** The author reported the result of a case shown by him at the Edinburgh meeting of the Society last year. The exophthalmos had now entirely disappeared, the bruit was no longer heard, and the vision now practically $\frac{6}{18}$ in each eye. A month after the carotid had been tied, and while the patient was still in hospital, he developed thrombosis of the left leg and thigh, but he made a good recovery from that and was discharged on August 2d, and had since resumed his work as a coachman. Early last month he came to see whether anything could be done to remedy the convergent strabismus which remained as a result of the paralysis of the right external rectus. The power of that muscle also had improved considerably, and Dr. Paterson tenotomized the internal rectus, leaving only 5 degrees of convergence. Dr. MACKAY also read a paper on a case of **phylctenular keratitis and pustular episcleritis treated by antistaphylococcic injections.** The patient at the age of 14 had keratitis in the left eye, with considerable impairment of vision, and five years later the other eye was affected, and she was under the care of Dr. Dufour, of Lausanne. His treatment was by hot fomentations, and anti-rheumatic powders internally. Shortly afterwards she consulted Dr. Mackay, and he found a small pustule on the right conjunctiva, which had discharged its contents and appeared as a raised ulcer. There was a haze in the cornea and a thinning in the ciliary region, but no pain. Hypermetropic astigmatism was present in both eyes, but she had not worn glasses. He ordered weak yellow oxide of mercury ointment, with atropin, and gray powder and bicarbonate of soda internally, followed by an arsenic and iron tonic. Under this she gradually improved, and correcting glasses were ordered. Three years later she returned with a small patch of injection in the edge of the right cornea, and there was a fringe of older opacity in the position of the arcus senilis. The same treatment again produced improvement. Subsequently there were several recurrences, and in August, 1906, he suspected the cause was tubercle, especially as her parents had been related before marriage, and she had some phthisical relations. Dr. Ian Stewart, at his request, took the

opsonic index, which was found to be for tubercle 0.74, and for staphylococcus aureus, 1.24. Films from the conjunctival sac showed large numbers of polymorphonuclear leucocytes and a few staphylococci, but no tubercle bacilli. Dr. Stewart thought the condition was not tuberculous. After a week, antistaphylococcic treatment was started, $\frac{1}{6}mg$ of dried staphylococcus aureus being given dissolved in 1*cc* of distilled water. The following day there was a decided negative phase, the index falling to 0.92, but 24 hours later it had risen to 1.86. Steady improvement set in, and in January this year she said she was very well and had kept so. The general aspect of her eye was much improved. He proceeded to discuss the ætiology of the episcleritis, and to bear testimony to the great value of the opsonic index, particularly in distinguishing tuberculous from staphylococcic infection. He laid stress on studying the positive or negative phase 24 hours after an injection. He believed the first suggestion of diagnosis by giving small doses of dead bacilli was made by Drs. Lawson of Banchory and Ian Stewart of Edinburgh, in the Edinburgh *Medico-Chirurgical Transactions*, 1905. Mr. JESSOP discussed the case, and Dr. MACKAY replied.

An ordinary meeting of the Society was held in the rooms of the Medical Society on Thursday June 11th, Mr. MARCUS GUNN, F. R. S. C., President, in the chair.

Mr. PRIESTLEY SMITH showed a small instrument, consisting of a series of lenses arranged in a frame, for use with the shadow test. The feature of the apparatus was the close packing of the lenses, about 16 of them being contained in a linear foot, and it enabled very rapid examination to be made. Mr. LESLIE PATON showed a case with connective tissue in front of the optic disk. Dr. EDRIDGE GREEN exhibited an **improved lantern for testing color-perception.** There was an arrangement consisting of three slides in a vertical column, each slide being fitted with variously colored glasses, which could be brought opposite an aperture with an iris diaphragm. Mr. J. H. TOMLINSON showed a Nernst-lamp projecting lantern. Mr. SYDNEY STEPHENSON showed a patient with a curious congenital anomaly of the iris. Mr. G. W. ROLL

showed a patient with sky-blue sclerotics, and Mr. G. W. ROLL one with double papillitis. Mr. HUDSON showed a case of Tiersch grafting. Mr. C. COATS showed a case of exudation in the retina. Mr. P. E. H. ADAMS read a paper on **An epidemic of contagious conjunctivitis due to the pneumococcus.** He said the epidemic occurred in the spring, in a small school of 37 boys, 21 of whom were attacked. The eldest boy affected was 14, the youngest nearly 10. All the smears taken, except one, showed numerous Gram-positive diplococci. Most of them were typically flame-shaped pneumococci with capsules, though a few of them were rounder in shape, and in some the capsules were not evident. This agreed with the description given by Schmidt (ARCH. OF OPHTH., xxxvii., No. 2) of smears taken from cases of pneumococcal conjunctivitis. On blood agar the colonies were typical slow-growing pneumococci, and there was no growth on agar. Only three of the cases were at all acute, most of them being subacute or quite mild. Four cases of relapse were reported. The condition found on examination was fairly constant: general congestion of the lower palpebral conjunctiva, with slight injection of the ocular conjunctiva, and in the acute cases a thin line of muco-pus in the folds of the conjunctiva of the lower lid. The upper lid showed slight enlargement of follicles at the angles, and a row of minute follicles along the everted edge. The whole epidemic lasted about five weeks, and spread in spite of isolation. It agreed in almost every respect with previously reported outbreaks, the chief difference being the generally mild nature of the cases.

Mr. NETTLESHIP read notes of three cases of **hereditary eye diseases.** Case 1 was that of a husband and wife who had senile cataract at an advanced age, and both were operated on with success. They had 10 children, and 7 had cataract in various degrees, 2 of these having had them "matured" and operated upon at a much earlier age than in their parents. There were also slight lenticular changes in the grandchildren of the original couple. The second family showed the occurrence of "Coppock" or discoid cataract, and lamellar cataracts of various sizes, and likewise of retinitis pigmentosa. Among about 250 persons the author had been able to trace,

belonging to the family, 32 with family cataract, and 15 with retinitis pigmentosa. The sex, incidence, and mode of descent were shown to be different for the cataract and retinal disease respectively. The third case was that of a family containing color-blind individuals. Mr. Nettleship had examined all the available members; and of 33 males 14 were found to be color-blind: and of 36 females only one was affected, and she slightly. There were 40 others in the pedigree who were either dead or inaccessible.

Lieut.-Col. H. HERBERT read a note on **secondary (parenchymatous) xerosis.** He drew attention to the part played by pannus in the development of xerosis after trachoma, and pointed out that old pannus rendered the corneal surface insensitive. Hence with both corneas covered with pannus, the blinking movements of the lids tended to be imperfect, and thus led to exposure of the globe. This appeared to be an important factor in the dryness which followed: for in such cases xerosis of the cornea and conjunctiva was often very marked when the cicatricial degeneration of the conjunctiva was much less advanced than in other old trachomatous cases in which the eyes remained moist and the corneas clear. The exposure of the cornea rendered it more opaque and insensitive, so that the blinking movements might amount to only very infrequent and slight twitches of the lids. Similarly inactive lids were seen in India in a few cases of severe primary (epithelial) xerosis. In these cases the exposed cornea became not only opaque, but pigmented, like the degenerate conjunctiva. Here the primarily defective reflex action of the lids seemed to be attributable to malnutrition only. Another factor in the production of cicatricial xerosis was the reduced transudation of fluid from the palpebral conjunctiva. If the upper lid were everted in any case of ordinary acute conjunctivitis, a free flow of watery fluid was readily demonstrable from the exposed conjunctiva. But a similar flow was not obtainable from a cirrhosed or cicatricial conjunctiva, however much inflammation might be present. Lieut.-Col. HERBERT also read a paper on **lower corneal plaques.** These, he said, were slightly raised, feebly vascular, gray patches with rough surfaces, close to the lower margin of the cornea. Their opacity varied with their thickness. They

were often of curious shapes, and were mostly multiple and binocular. They were not very uncommon in India, and appeared to be due to slightly defective closure of the lids, together with more or less chronic conjunctivitis, trachomatous or otherwise.

An ordinary meeting of the Society was held in the rooms of the Medical Society on Friday, July 10th, Mr. MARCUS GUNN, F.R.C.S., President, in the chair.

Mr. W. H. JESSOP showed a case of **pseudoglioma**, and a beautiful series of photographs of the **fundus oculi**, by Professor Dimmer. Electricity was the illuminant. Mr. Bardsley showed several ingenious mechanical improvements in instruments. One was a new form of needle-holder, another an adjustable modified trial frame, and a third an improvement in the "Moorfields" ophthalmoscope, introducing a double hole for indirect vision.

Mr. M. L. HEPBURN read a paper, with lantern illustrations, on **the visual fields in pigmentary degeneration of the retina.** The contribution was based upon 26 cases, which were tested in various ways, owing to the risk of error, and the knowledge that much depended on the intelligence of the patient. His chief concern had been to estimate the amount of relatively functioning area remaining—in other words, how much of the retina had escaped destruction in such cases. Unless the rods and cones had been hopelessly damaged, the retention of visual acuteness was a matter of degree. Where the patient showed uncertainty, the matter could be placed beyond doubt by stronger illumination. As it was desirable to have a definite standard to work to, he had taken as his standard a 10-millimetre test object in natural light. He pointed out the relationship between the several varieties, and showed slides in illustration. The pathological changes in chronic inflammation of the choroid were of two kinds, and were represented by two kinds of visual fields. He raised the question whether the islands of vision occasionally seen through scotomata represented portions of retina which had escaped disease, or did they represent the re-establishment of the circulation, or a new system of vessels.

SYSTEMATIC REPORT ON THE PROGRESS OF OPHTHALMOLOGY IN THE THIRD AND FOURTH QUARTERS OF THE YEAR 1907.

By Dr. G. ABELSDORFF, in Berlin; Prof. ST. BERNHEIMER, in Innsbruck; Dr. O. BRECHT, Prof. R. GREEFF, Prof. C. HORSTMANN, and Dr. R. SCHWEIGGER, in Berlin; with the Assistance of Prof. A. ALLING, New Haven; Prof. E. BERGER, Paris; Prof. CIRINCIONE, Genoa; Dr. DALÉN, Stockholm; Prof. HIRSCHMANN, Charcow; Dr. J. JITTA, Amsterdam; Mr. C. DEVEREUX MARSHALL, London; Dr. H. MEYER, Brandenburg; Dr. P. VON MITTELSTÄDT, Metz; Dr. H. SCHULZ, Berlin; Prof. DA GAMA PINTO, Lisbon; and Others.

Translated by Dr. PERCY FRIDENBERG, New York.

Sections VIII.–XII. Reviewed by DR. R. SCHWEIGGER, Berlin.

VIII.—LIDS.

266. CHERNO, FRANZ. **On blepharitis ciliaris, so-called.** *Zeitschr. f. Augenheilk.*, xviii., No. 1, p. 1.

267. NATHANSON, A. **Chronic multiple suppuration of Meibomian glands.** *Klin. Monatsbl. f. Augenheilk.*, xlv., No. 2, p. 529.

268. LEWANDOWSKY, M. **Loss of function of lid closure.** *Berliner klin. Wochenschrift*, 1907, No. 29.

269. MOTAIS. *New Data on the Motais Operation.* Angers, 1907.

270. STRAUB, M. **Entropium operation.** *Nederl. Tydschrift voor Geneesk.*, 1907, Bd. ii., No. 11.

271. MAY, C. H., and HOLDEN, W. A. **Case of upper-lid cyst with microphthalmus.** *Arch. f. Augenheilk.*, lviii., No. 1, p. 43.

272. RAMPOLDI, R. **Therapeutic action of jequirity in some cases of cancer.** *Annali di ottalm.*, xxxvi., Nos. 3–4, p. 296.

273. CLAIBORNE, J. H. **Case of angiosarcoma of the lower lid, with microscopic slide and photograph.** *The Ophthal. Record*, June, 1907.

274. ALLING, A. N. **Primary sarcoma of the eyelid.** *The Ophthal. Record*, June, 1907.

275. POSEY, W. C. **Coloboma of the lid, with anomalous con-**

dition of the tissues at the outer commissures. *The Ophthal. Record,* April, 1907.

CHERNO (266, **On blepharitis ciliaris, so-called**) recognizes three forms of lashes: (1) Long, evenly curved, and not brittle hairs, with cylindrical root-portion, the full-grown normal cilia; (2) shorter, thick, straight, and brittle hairs with a button-like root portion, immature form; and (3) a diseased form which never develops into cylindrical cilia, which he calls belated button-hairs. These last, alone, are to be epilated invariably. Infection generally takes place at the change of cilia. The various forms of blepharitis are considered. Sycosis and eczema are common. Acarus mites were often found in normal and diseased lashes of old patients, but never in children.

NATHANSON (267, **Chronic multiple suppuration of Meibomian glands**) reports four cases without involvement of the conjunctiva, swelling and œdema of the lids, and with no tendency to spontaneous perforation. The process tends to creep along in the depth. There was some nasal disease in three cases and ozæna in one. Staphylococci were found in the pus from one case. N. calls the disease chronic suppurative Meibomian adenitis poly.

LEWANDOWSKY (268, **Loss of function of lid closure**) observed loss of active lid closure in an old man, coming on after hemiplegia and remaining permanently. The patient was unable to close either eye or both eyes at command, but there was no interference with involuntary winking.

MOTAIS (269, **New data on the Motais operation**) reports a number of cases to show the good results obtained with his method, both in congenital (Bassa, Gendron, Truc, Lagrange) and acquired (Trantas, Dickson-Bruns) cases. His last modification consists in the excision of a piece of skin from the upper lid in cases of thickening of the tissues, as in diffuse lipoma, chronic œdema, old trachoma, or essential shrinking of the conjunctiva, which interfere with the mobility of the upper lid. BERGER.

STRAUB (270, **Entropium operation**) lays stress on the cicatricial retraction in the conjunctiva of the upper part of the tarsus. The traction affects the position of the lid margin,

but there is no independent deformation of the tarsus, and its excision is rarely required, exsection of the muscle fibres and of the cicatricial strands, with direct attachment of the skin to the cartilage, being generally sufficient.

May (271, **Case of upper-lid cyst with microphthalmus**) describes a congenital hollow tumor connected with a rudimentary eye, almost the size of the normal fellow, but with microcornea and cataracta accreta. Rosettes were found in the invagination of the secondary optic vesicle lining the cyst, by Holden, who shows that these figures depend on a typical development of embryonic cells which would otherwise form rods and cones.

Rampoldi (272, **Therapeutic action of jequirity in some cases of cancer**) reports two cases of carcinoma of the lid cured by the use of jequirity-gelatine disks, as prepared and sold by the chemist Leopold Zambeletti in Milan.

Calderaro.

Claiborne's (273, **Case of angio-sarcoma of the lower lid, with microscopic slide and photograph**) patient with angiosarcoma of the lower lid was a boy of 13 years, who showed a small, red, painless, pedunculated tumor on the right lower lid near the inner angle. Sarcoma cells were found in sections. The growth was excised, but grew rapidly and was destroyed with the cautery. Blok in 1905 reported an angiosarcoma of the lid. Blake.

Alling's (274, **Primary sarcoma of the eyelid**) patient with sarcoma of the lid was a girl of 7 years in whose left lower eyelid a tumor had been present for about six weeks. A complete dissection was made, but recurrence took place in two months, when a more radical operation was done. The child died of intercurrent affection shortly afterward. The growth was a fibro-sarcoma, non-pigmented. The writer finds about 65 cases reported to date.

Posey (275, **Coloboma of the lid, with anomalous condition of the tissues at the outer commissures**) saw a congenital coloboma of the left upper lid. It was situated in the median portion and involved the entire thickness of the lid. In the outer commissure of both eyes there was a thickening of the capsule and fascia, covered by the bulbar conjunctiva.

Blake.

IX.—LACHRYMAL APPARATUS.

276. Dor, H. **Cyst of lachrymal gland.** *Rev. gén. d'opht.*, xxvi., p. 337.

277. Gutmann, Ad. **Mikulicz's disease and its relation to lues.** *Berliner klin. Wochenschrift*, 1907, No. 38, p. 1141.

278. Napp, O. **Relation of Mikulicz's disease to tuberculosis.** *Zeitschrift f. Augenheilk.*, xvii., No. 6, p. 513.

279. Freund. **Case of Mikulicz's disease treated with X-rays.** *Deutsche med. Wochenschrift*, 1907, No. 38, p. 1560.

280. Koster, Gzn., and Kan, P. **New treatment in chronic lachrymal disease.** *Nederl. Tydschrift voor Geneesk.*, Bd. ii., No. 10, 1907.

281. Villard, H. **Optic nerve atrophy following acute dacryocystitis.** *Annales d'oculistique*, cxxxviii., No. 34, p. 24.

282. Wirtz, R. **Clinical and pathological study of a case of primary tuberculosis of the lachrymal sac.** *Klin. Monatsbl. f. Augenheilk.*, xlv., No. 1, p. 523.

Dor (276, **Cyst of lachrymal gland**) reports the case of a child of 3½ months, with an orbital tumor forcing the globe outwards. On extirpation it was found to be a cystic retention tumor, probably congenital, of the lachrymal gland. The youth of the patient is the rare feature of the case. In the work of Lagrange (Paris, 1904), 23 cases are reviewed, the youngest being 6 years old.

Gutmann (277, **Mikulicz's disease and its relation to lues**) observed a case of Mikulicz's disease with marked, painless swelling of lachrymal, parotid, submaxillary, and sublingual glands on both sides, depending on lues of 3 years' standing. Large doses of iodides relieved all the symptoms, including a follicular conjunctivitis, in 3 months. The diagnosis of syphilis in other cases reported is not proven.

Napp (278, **Relation of Mikulicz's disease to tuberculosis**) believes that Mikulicz's disease is not uniform, but rather a symptom-complex, which may present as an isolated affection of lachrymal and salivary glands, of lachrymal glands alone, or as disease of the remaining lymphadenoid structures. Chronic inflammatory changes on a basis of tuberculosis, with induration in the stage of healing, explain the local tissue changes. Yellowish-gray nodules, from the size of a millet seed to that of a pea, were seen in the mucous membrane of the mouth and conjunctiva. There was no ulceration.

Microscopic examination showed that these nodules were tubercles containing bacilli.

KOSTER (280, **New treatment in chronic lachrymal disease**) uses a silk thread which is introduced by means of a hollow de Wecker sound and threaded into the nose. The patient can be taught to remove and reintroduce the twisted thread probe from time to time, and it may remain *in situ* for several months.

VILLARD (281, **Optic nerve atrophy following acute dacryocystitis**) saw the development of optic nerve atrophy after acute dacryocystitis of the same side, in a man of 56. Seventeen cases of this sort are recorded in the literature. In Villard's case the dacryocystitis had been treated in the usual way with incision and antisepsis. BERGER.

WIRTZ (282, **Clinical and pathological study of a case of primary tuberculosis of the lachrymal sac**) recommends a careful test by tuberculin injections, animal experimentation, and examination for bacilli, in marked, circumscribed swelling of the sac, without much pus production, particularly in youthful subjects. In tissue sections it is often difficult to distinguish old tubercular foci from simple granulation tumors and sarcoma. In this case there was no other focus of tuberculosis, but the marked reaction to tuberculin and the integrity of the epithelium of the sac spoke strongly in favor of endogenous infection.

X.—ORBIT AND ACCESSORY CAVITIES.

283. UHTHOFF. **Orbital hemorrhage with exophthalmos after pertussis.** *Berliner klin. Wochenschrift*, 1907, No. 45, p. 1466.

284. TAVEL. **Operative technic in extirpation of retrobulbar tumors.** *Deutsche Zeitschrift f. Chirurg.*, lxxxvii., p. 561.

285. BECKER, (COBLENZ). **Traumatic aneurysm of internal (cerebral) carotid with pulsating exophthalmos.** *Centralbl. f. Chirurg.*, 1907, No. 31, p. 13.

286. PINCUS, FR. **Spontaneous cure of traumatic pulsating exophthalmos.** *Zeitschrift f. Augenheilk.*, xviii., No. 1, p. 33.

287. MONTHUS and CARISE, A. **Orbital varicocele.** *Soc. d'opht. de Paris*, vi., No. 4, 1907.

288. CASALI, A. **Mucocele of right frontal sinus.** *Ann. di ottalm.*, xxxvi., No. 304, p. 267.

289. CUNNINGHAM, H. **Empyema of sphenoid sinus with ocular symptoms.** *Zeitschrift f. Augenheilk.*, xvii., No. 4, p. 345.

290. COPPEZ, H. **Two cases of blindness due to sphenoid-sinus disease.** *Journ. de méd. de Bruxelles*, v., No. 15, 1906.

291. STOEWER, P. **Treatment of septic thrombosis of cavernous sinus of orbital origin.** *Klin. Monatsbl. f. Augenheilk.*, xlv., No. 2, p. 147.

292. BIRCH-HIRSCHFELD, A. **Optic-nerve affections in disease of the posterior accessory cavities of the nose.** *v. Graefe's Archiv f. Ophth.*, xlv., No. 3, p. 440.

293. ONODI, A. *The Optic Nerve and the Accessory Cavities of the Nose.* Vienna and Leipzig, 1907.

294. HARRISON BUTLER, T. **A case of Chloroma.** *British Medical Journal*, April 20, 1907.

295. TOOKE, F. T. **Hemorrhages within the orbit, with report of a case of spontaneous origin.** *The Ophthal. Record*, April, 1907.

296. HENDERSON, F. L. **Thrombosis of the cavernous sinus.** *Amer. Jour. of Ophthal.*, March, 1907.

297. PARKER, W. R. **Neuro-fibroma of the orbit. Krönlein's operation.** *Jour. Amer. Med. Asso.*, July 6, 1907.

298. PECK, W. H. **Unilateral exophthalmos in a new-born infant.** *Annals of Ophth.*, July, 1907.

299. LEENHEER, C. A. **An orbital tumor of ten years' standing.** *The Ophthal. Record*, May, 1907.

UHTHOFF (283, **Orbital hemorrhage with exophthalmos after pertussis**) treated a boy of 4 for protrusion of both eyes coming on with attacks of whooping-cough. On the right side the orbital region gradually protruded, with final pulsating exophthalmos. Probatory incision evacuated a cyst containing a great deal of tarry blood. The eyeball then returned to its normal position, and complete recovery followed.

TAVEL (284, **Operative technic in extirpation of retrobulbar tumors**) performs Kroenlein's osteoplastic resection with a modification designed to avoid injuring the temporal branches of the facial nerve. He makes an incision 1*cm* long at the base of the frontal process of the zygoma and chisels through the bone into the inferior orbital fissure, from this point. A second cut is made through the brow from the inner end to the outer, and prolonged toward the zygoma, nearly reaching the articular tubercle. From this point the external orbital process is chiselled through parallel to the zygoma in the direction of the optic foramen. The large half-moon-shaped skin flap thus produced is then turned down with the loosened external wall of the bony orbit, giving free access to the depths.

BECKER'S (285, **Traumatic aneurysm of internal (cerebral)**

carotid with pulsating exophthalmos) patient had been injured by the explosion of a gun. Ligation of the internal carotid caused immediate reduction, but was followed by somnolence and recurrence of pulsation. Continued pressure, 8 hours daily, and ice-bags applied to the eye, for a space of 5 months, brought about a complete cure.

Pincus (286, **Spontaneous cure of traumatic pulsating exophthalmos**) reports spontaneous recovery from pulsating exophthalmos. The patient, a young man, had become blind in the right eye after a fall on the right side of the head. Six weeks later right-sided headache and exophthalmos came on with increase of objective noises on the left side. Digital compression of the left carotid had no effect, but right compression stopped the bruit, but at the beginning caused paresis and twitching of left arm, vertigo, and slight disturbance of speech. (The patient was left-handed.) Repeated compression of the carotid by the patient caused a disappearance of the left exophthalmos, but this now appeared on the right side. An ulcer on the somewhat anæsthetic right cornea, without discoverable cause, gradually healed. After ten years, marked headache developed, and while the patient was compressing the carotid the symptoms on the left side suddenly disappeared completely. Pincus thinks that the orbital veins were torn on the right side, the vessels on the left side being dilated by the aneurysm of the carotid in the cavernous sinus, and that thrombosis developing with intense headache finally stopped the pulsation and symptoms due to it.

Casali's (288, **Mucocele of right frontal sinus**) case of chronic inflammation of the frontal sinus followed injury to the right brow and caused retention in an accessory cell. The communication between the two cavities was shut off and the contents of the cell degenerated to a yellowish sirupy fluid containing cholesterin. This cyst was evacuated by incision above the brow. Its posterior wall was elastic and pulsated with the meningeal arteries in consequence of complete pressure atrophy of both bony plates. Calderaro.

Cunningham (289, **Empyema of sphenoid sinus with ocular symptoms**) reports a case of empyema of the sphenoid sinus with relapsing diplopia, fetid discharge from the left nostril,

and pain in the left brow and temple. Slight protrusion of the left eye and mild papillitis without marked disturbance of vision developed. This all cleared up after evacuation of thick pus from the sphenoid sinus, and repeated irrigation. Exophthalmos was probably due to direct extension of the sinus inflammation to the thin walls of the orbit. The sinus may be so eccentric as to cause inflammation of the contralateral orbit and optic nerve.

COPPEZ (290, **Two cases of blindness due to sphenoid-sinus disease**) reports two cases of blindness following sphenoidal sinusitis. In one, that of a woman of 58, influenza was the cause. Curettage and drainage of the sinus was followed by improvement of vision in the opposite eye, but the eye of the same side had lost all vision in consequence of optic-nerve atrophy, and was not improved. The sphenoid sinus was reached by Kroenlein's osteoplastic resection of the outer orbital wall. In a second case, no pus was found in the sinus, and the ocular symptoms remained unchanged.

STOEWER (291, **Treatment of septic thrombosis of cavernous sinus of orbital origin**) calls attention to the possibility of a spreading of infection from the orbit by thrombosis of small veins, direct extension through the orbital walls, and by anastomoses of the anterior facial vein with the superior ophthalmic, in purulent affections of the skin of the face. Prophylactic ligation of the angular vein might be of value in commencing thrombosis. Immune sera for streptococci, and so on, are not strong enough to be of service. Incisions of the orbital cellulitis are of no use after septic thrombosis has become established. The sinus may be opened according to Knapp, or free incisions made into the large orbital veins, after operation according to Kroenlein.

BIRCH-HIRSCHFELD (292, **Optic-nerve affections in disease of the posterior accessory cavities of the nose**) notes that blindness may come on rapidly at a very early stage. Central scotoma may be the first symptom.

ONODI (293, **The optic nerve and accessory cavities of the nose**) discusses the topographic anatomy of the orbit in relation to the accessory cavities in a monograph which is richly illustrated.

HARRISON BUTLER (294, **A case of chloroma**) gives a de-

tailed account of a case of chloroma, the 27th to be recorded, which he had under his care.

L. C., female, aged 11; was admitted to the Coventry Hospital on Dec. 20, 1906, suffering from exophthalmos and anæmia.

Condition on Jan. 1, 1907: Great proptosis of right eye, which is displaced outwards and downwards. Marked exophthalmos on the left side. Much engorgement of the conjunctival veins, and of those of the eyelids and temples. Chemosis of the right conjunctiva. Each disk is choked. Rt. +2.5 D.; Lt. +1.5 D. Small retinal hemorrhage. V R = $\frac{6}{9}$; T R = $\frac{1}{2}$; V L = $\frac{6}{9}$; Tn. Each temporal fossa is bulged out by a semifluctuating swelling. The child is very ill. Her skin is waxy and she is very anæmic. She can walk, and is quite intelligent. General condition normal, no enlarged glands, no subcutaneous hemorrhages.

Blood: Red cells, 3,000,000.
White cells, 26,000.

Differential count:

Polymorphonuclear	2.
Large lymphocytes	69.
Large mononuclear	14.8
Small lymphocytes	14.2
Eosinophiles, Mast cells	absent

Progress of case:

Jan. 10th.—Purpuric hemorrhages on buttocks and thigh. Saliva blood-stained. Patient is *deaf* on the right side.

Jan. 24th.—Alarming hemorrhage from the orbit, at the end of which the eye sank back into a normal position.

Jan. 30th.—Increased proptosis of the right eye. The lower cul-de-sac is occupied by a bleeding, fungating mass which, however, shows no green color. The right cornea has sloughed. Right ear very deaf.

Feb. 17th.—Dyspnœa, semi-comatose condition.

Blood: Red cells, 1,450,000.
White cells, 17,000.

Child died on Feb. 18th, the case having been previously diagnosed as "*chloroma.*"

Post-mortem: On removing *sternum* a large flat growth

was seen to cover its inner side; it was olive green in color, and lay under the periosteum. The *ribs* were green by transmitted light. There was a pint of blood-stained fluid in each *pleural cavity*. At the apex of each lung was a mass of green growth. The *spine* along its whole length was covered by masses of green growth up to ¾ inch thick, which in the thorax was continued along the ribs. There was a congenital narrowing of the first four inches of the ascending colon. Both *ovaries* enormously enlarged and infiltrated with a green growth.

The right *eye* was disorganized. There was only a small mass of tumor deep in the orbit. The *lachrymal gland* was infiltrated and green. There was a mass of growth in the *temporal fossa*. Growth along the *sagittal* and *coronal sutures* and on the *occipital bone*. *Meninges* infiltrated.

The right *cavernous sinus* was solid with green growth, and the *lateral sinus* was occluded.

After twenty-four hours in 10% formol the green color was still visible. The tumor was *a round celled sarcoma* with unusually well marked stroma.

The case lasted seven weeks. The exophthalmos was due to vascular engorgement; it disappeared after death.

A very similar case was recorded by Dunlop in the *Brit. Med. Journal*, May 3, 1902.

The specimens from this case are preserved in the Museum of St. Bartholomew's Hospital.

T. HARRISON BUTLER.

TOOKE (295, **Hemorrhages within the orbit, with report of a case of spontaneous origin**) could find no cause, either in the history or physical condition of his patient, to account for the occurrence of hemorrhage. A partial atrophy of the optic nerve resulted. BLAKE.

Facial erysipelas led in HENDERSON'S (296, **Thrombosis of the cavernous sinus**) case to an orbital cellulitis. Deep incision disclosed no pus, and the symptoms later pointed to sinus thrombosis. Exposure of the lateral sinus showed pus and clotted blood. At the post-mortem there was found suppuration of the lateral cavernous and inferior petrosal sinuses, and a clot extending into the jugular vein.

According to PARKER (297, **Neuro-fibroma of the orbit.**

Krönlein operation) there are but two cases on record of this variety of tumor of the orbit. His case gave a history of exophthalmos increasing during twelve years. The tumor removed by Krönlein operation measured 35 x 25 x 9*mm.* It apparently had its origin in the sheath of the frontal branch of the ophthalmic at the junction of the supratrochlear and supraorbital nerves. It consisted of fibrous connective tissue arranged in coarse and fine fibrils. Running through the mass are nerve fibrils in part connected as a nerve trunk but for the most part separated by connective tissue. The diagnosis was œdematous neuro-fibroma or fibromatosis of the nerve trunk. ALLING.

PECK'S (298, **Unilateral exophthalmos in a new-born infant**) case showed no bruit or pulsation and the child had not been delivered with instruments. The exophthalmos became gradually more pronounced for a week so that the cornea was excoriated from the inability to close the lids. The eye then gradually receded and finally became absolutely normal. The author suggests that some vascular disturbance may have been the cause and that it was relieved later by collateral circulation. ALLING.

The usual symptoms of orbital tumor of ten years' standing were present, including optic nerve atrophy, in LEENHEER'S (299, **An orbital tumor of ten years' standing**) patient. The Krönlein operation was done but the growth recurred shortly. There were no evidences of metastases. The tumor presented the microscopic characters of a hemangio-endothelioma, evidently of no great malignancy. BLAKE.

XI.—CONJUNCTIVA.

300. CAUER. **Two cases of indirect rupture of the upper tarsus.** *Klin. Monatsbl. f. Augenheilk.*, xlv., No. 2, p. 280.

301. TROUSSEAU, A. **Conjunctivitis with adenopathy.** *Annales d'oculistique*, cxxxvii., p. 449.

302. GREEFF, R. **Peculiar double granules (parasites?) in trachoma cells.** *Deutsche med. Wochenschrift*, 1907, No. 27, p. 914.

303. GREEFF, R. **Parasitary cell-inclusions in trachoma.** *Ibid.*, No. 27, p. 1104.

304. GREEFF, R. **Correction to above.** *Ibid.*, No. 32, p. 1302.

305. GREEFF, FROSCH, and CLAUSEN. **Studies in etiology and development of trachoma.** *Archiv f. Augenheilk.*, lviii., No. 11, p. 52.

306. HALBERSTAEDTER and PROWAZEK. **Etiology of trachoma.** *Deutsche med. Wochenschrift*, 1907, No. 32.

307. ORLOW, K. **Pathologic anatomy of trachoma.** *Westn. Ophthalm.*, 1907, No. 5.

308. NICOLLE and CUENOD. **Experimental reproduction of granular conjunctivitis in monkeys.** *Acad. des Sciences*, Paris, v., No. 6, 1907.

309. NICOLLE and CUENOD. **Idem.** *Muench. med. Wochenschrift*, 1907, No. 28, p. 1412.

310. FERMI, CLAUDIO, and REPETTO, R. **Filtration of trachoma germs and significance of cultivable flora of conjunctiva.** *Berliner klin. Wochenschrift*, 1907, No. 38, p. 1197.

311. BAJARDI. **Transmissibility of trachoma from man to apes.** *Clin. oculist.*, i., 1907, p. 2719.

312. AMBERG, H. **Conjunctival shrinking after pemphigus.** *Zeitschrift f. Augenheilk.*, xvii., No. 5, p. 417.

313. ADAMUECK, V. *Local amyloid of conjunctiva.* Kasan, 1907.

314. OATMANN. **Epithelial inclusion cysts of the conjunctiva.** ARCH. OF OPHTH., xxxvi., No. 3.

315. COSMETTATOS, G. **Epithelial cysts of conjunctiva.** *Archives d'ophtalm.*, xxvii., p. 236.

316. ADAMUECK, V. **Double tarsus of the lid.** *Westn. Ophthalm.*, 1907, Nos. 3 and 4.

317. DEBEVE, J. **Primary epithelioma of bulbar conjunctiva.** *Arch. d'opht.*, xxvii., p. 478.

318. v. SICHERER, O. **Epibulbar pigmented sarcoma.** *Zeitschrift f. Augenheilk.*, xvii., No. 5, p. 428.

319. BARLINI, C. **Clinical and anatomical study of lymphoma of conjunctiva and tarsus.** *Clin. oculist.*, v., 1907, p. 2850.

320. STEPHENSON, S., and FORD, ROSA. **Ante-partum ophthalmia.** *Ophthalmoscope*, April, 1906.

321. FORD, ROSA. **A further note on ante-partum ophthalmia.** *Ophthalmoscope*, October, 1906, and May, 1907

322. ROBINSON, G. D. **Ante-partum ophthalmia; three cases.** *Ophthalmoscope*, June, 1907.

323. MANCHÉ, C. **Some notes upon granular conjunctivitis or trachoma.** *Ophthalmoscope*, August, 1907.

324. ELLIOT, R. H. **Conjunctival asthenopia due to glare.** *Ophthalmoscope*, Feb., 1907.

325. HARRISON BUTLER, T. **The clinical features, bacteriology, and treatment of acute ophthalmia in the East.** *Royal Lond. Ophth. Hosp. Reports*, March, 1907.

326. MCKEE, H. **A new pathogenic micro-organism of the conjunctival sac.** *Jour. Med. Research*, July, 1907.

327. CRISP, J. D. **Chancroidal conjunctivitis.** *Denver Med. Times*, July, 1907.

328. JOHNSTON, R. H. **Pseudo-membranous conjunctivitis caused by staphylococcus aureus and albus.** *The Ophthal. Record*, June, 1907.

329. KRAUSS, F. **Parinaud's conjunctivitis; a report of two cases.** *Annals of Ophthal.*, January, 1907

330. WOOD, C. A. **The unilateral form of tarso-palpebral vernal conjunctivitis occurring in a young girl.** *The Oph. Record*, June, 1907.

331. PUSEY, B. **Involvement of the cornea and bulbar conjunctiva in the secondary stage of syphilis.** *Jour. Amer. Med. Asso.*, September 7, 1907.

CAUER (300, **Two cases of indirect rupture of the upper tarsus**) noted this injury in two cases after blow on the lid with a blunt instrument. It was probably caused by violent stretching of the lid, and produced a vertical tear.

TROUSSEAU (301, **Conjunctivitis with adenopathy**) noted this condition limited to one side and mainly affecting the outer angle, in a patient of fifteen. There was enormous swelling of the preauricular, parotid, and sub-maxillary glands. Bacteriologic examination, negative. Complete spontaneous recovery in one month. Possibly Parinaud's disease.

GREEFF (302, **Peculiar double granules—parasites?—in trachoma cells**) found small double granules as cell inclusions in trachoma which are much smaller than cocci and may appear outside of the follicles.

GREEFF, FROSCH, and CLAUSEN (305, **Studies in etiology and development of trachoma**) looked for spirochæte in the secretion and follicles of trachoma. The cells of trachoma bodies are not phagocytic, but are epithelial derivates. Small diplococci surrounded by an areola are often found about the nucleus.

HALBERSTAEDTER and PROWAZEK (306, **Etiology of trachoma**) show cell inclusions which could be inoculated into apes. They were not found in other conjunctival affections. They are analogous to the germs of small-pox, cow-pox, scarlet fever, epithelioma of fowls, and molluscum contagiosum, and constitute a special class, chlamydozoa.

ORLOW (307, **Pathologic anatomy of trachoma**) subjected undoubted trachoma follicles, in eighteen cases, to careful microscopic examination with the stains of Mallory, Unna, and others. Elective staining after Feodorow of follicles previously digested with trypsin showed a connective-tissue framework and blood vessels in trachoma follicles as well as in normal follicles from the conjunctiva, in young and

developed follicles, consisting of a tunica propria and endothelium. These structures are lymph-follicles *sui generis*, not granulomata (Parisotti).

HIRSCHMANN.

NICOLLE and CUENOD (308 and 309, **Experimental reproduction of granular conjunctivitis in monkeys**) were able to inoculate apes (*Macacus sinicus*) with curettings from trachoma follicles, as Hess had previously done. Thirty-six days later, histological examination of excised pieces of conjunctiva showed complete identity of structure of trachoma granules of apes with those of human beings.

BERGER.

In a preliminary communication FERMI and REPETTO (310, **Filtration of trachoma germs and significance of cultivable flora of conjunctiva**) report on flora of the conjunctival sac in trachoma, and conclude that none of the cultivable forms, either alone or together, are able to cause the clinical picture of trachoma. The specific virus may lose its virulence in process of cultivation.

BAJARDI'S (311, **Transmissibility of trachoma from man to apes**) inoculation experiments with trachoma were applied to apes. Fluid from active trachoma follicles was instilled into the leeched conjunctiva or injected. Positive results invariably followed in about four weeks. Injection of a filtrate was negative. CALDERARO.

AMBERG (312, **Conjunctival shrinking after pemphigus**) reports the case of a boy of seven, of healthy family, with chronic pemphigus, eruptions of which had caused symblepharon, entropion, complete obliteration of fornices, and cicatricial degeneration of the cornea. The shrinkage is not "essential," but a result of the bullæ, which, like herpes, produce no skin scars but leave deep ulcers in mucous membranes. Histologic examination showed chronic inflammation of the stroma of the conjunctiva, with shrinkage, and epithelial proliferation and horny degeneration.

ADAMUECK (313, **Local amyloid of conjunctiva**) discusses the history, geographical diffusion, statistics, and clinical picture of this affection, its relation to hyaline, results of microscopic examination, and the pathologic reactions. Amyloid is probably an interstitial deposit. Cellular elements show no amyloid de-

generation. The connective-tissue fibres become atrophied by pressure of the masses deposited between them. Vessels are narrowed and finally occluded, in the same way. Elastic fibres resist for a long time. Calcification and bone deposits are often found. Amyloid may be eliminated by giant cells or become encapsulated. Amyloid particles introduced under the conjunctiva or the skin or into the anterior chamber are gradually absorbed after setting up inflammatory reaction. Giant cells and polyblasts are active in this process. Trachoma and chronic inflammation are the most important etiologic factors. Micro-organisms could not be detected. HIRSCHMANN.

OATMANN (314, **Epithelial inclusion cysts of the conjunctiva**) noted the development of tense cysts in the transition folds after expression of trachoma. Clear fluid was evacuated on incision and quickly renewed. The walls are composed of fibrous connective tissue, covered on both sides with epithelium. They are probably due to islands of epithelium, remaining after expression, and to cohesion of surfaces during the stage of reactive swelling and inflammation. Epithelial proliferation of these inclusions then causes the formation of cysts.

TREUTLER.

COSMETTATOS (135, **Epithelial cysts of conjunctiva**) describes several cases of epithelial cysts of the conjunctiva. They may develop in Krause's glands, Henle's pseudo-glands, or from abnormal conjunctival folds in chronic conjunctivitis.

BERGER.

ADAMUECK (316, **Double tarsus of the lid**) describes a condition similar to that named "epitarsus" by Schapringer, consisting in a doubling or apron of the conjunctiva of the upper lid. Microscopic examination showed the structure of tarsal cartilage covered with normal conjunctiva.

HIRSCHMANN.

DEBEVE (317, **Primary epithelioma of bulbar conjunctiva**) observed the development of epithelioma of the conjunctiva bulbi in a man of forty. Examination of the enucleated globe showed that the new growth had invaded the cornea and sclera and had infiltrated the region of Schlemm's canal and Fontana's spaces. The anterior chamber would probably have been invaded before long. BERGER.

SICHERER (318, **Epibulbar pigmented sarcoma**) describes an epibulbar pigmented sarcoma which had developed from a nævus and in four years attained the size of an apple. Ulceration and painful metastases in the liver led to general cachexia and finally to death.

BARLINI (319, **Clinical and anatomical study of lymphoma of conjunctiva and tarsus**) reports the case of a boy with œdema of the left upper lid, ptosis, and large red granulations on the tarsal conjunctiva. Total extirpation of the tarsus and conjunctiva was followed by recovery of function of the upper lid, by means of the muscle fibres running from the levator to the skin of the lid. Histological examination showed the structure of lymphoma of the conjunctiva and tarsus. CALDERARO.

STEPHENSON and FORD (320, **Ante-partum ophthalmia**). The authors remark that cases in which babies are born with symptoms of ophthalmia are not so rare as has been supposed. They have collected thirty-seven cases from published records, and give an account of seventeen new cases. These cases both old and new are analyzed in tabular form. In the second and third papers Rosa Ford adds three more cases, and in the final communication Robinson gives an account of three more, bringing the total up to sixty cases in all. In patients inoculated with gonorrhœal pus for the cure of pannus, Bellonard (*Thèse de Paris*, 1892) noted an invariable interval of two and a half to three days before the eyes showed any sign of inflammation. The joint authors of the first paper analyze the cases under the following heads: time before birth when membranes ruptured; circumstances attending labor; condition of the child; state of the eyes; bacteriology; condition of the mother; course of the ophthalmia; and general remarks.

The authors conclude that: about half the cases can be accounted for by premature rupture of the membranes allowing micro-organisms to reach the baby's eyes. In the remainder the organism must either have entered through the unruptured membranes, or by a slight injury therein. Increased temperature of the conjunctiva in utero, enhanced virulence of the organisms, feebleness of the babies, tears of the membranes, position of the fœtus, and condition of the

placenta cannot be connected with the causation of ante-partum ophthalmia. Several of the so-called "congenital" anomalies of the eyes, as corneal opacities, staphyloma, microphthalmos, cryptophthalmos, and lachrymal abscess, are probably to be explained on the theory of an intrauterine infection.

In Rosa Ford's (321, **A further note on ante-partum ophthalmia**) second paper she describes a case upon which she thus comments: "We have to account for two obvious facts, viz., the occurrence of infection before labor commenced, it being certain that 8½ hours is an impossibly short time to allow for the incubation of the micro-organism together with the production of so great a degree of inflammation (the child was born with severe ophthalmia, labor lasted eight hours ten minutes, the membranes were ruptured artificially forty minutes before birth); and the early stage of the inflammation. That is, we must try to discover why, since infection occurred before labor, it should yet have been so near labor that the inflammation was still in its first stage." She suggests that the patulous condition of the external os may allow gonococci to enter the uterus. She believes that they entered in this manner and passed through the unbroken membranes. In the third paper Rosa Ford cites two more cases; in both, ophthalmia was present at birth. In each, labor was short, seven and ten hours, so that infection must have reached the eyes before labor began. The membranes had ruptured ten minutes and one hour before delivery, so the micro-organisms had apparently penetrated the unbroken membranes. She concludes that taking all the cases together it is practically certain that in many cases the eyes of the child can be and are infected whilst still within the unruptured membranes. Robinson records three very similar cases. All the babies he records were born with ophthalmia which became worse in spite of immediate treatment with nitrate of silver 2%.

T. Harrison Butler.

Manché (323, **Some notes upon granular conjunctivitis or trachoma**) of Malta gives a short account of trachoma based on discussions of the Italian National League against trachoma and of the Italian Ophthalmological Society in 1906.

Contagiousness.—Trachomatous material placed in the eyes of monkeys brings on in two or three weeks an acute conjunctivitis, which a month later presents all the characters of true trachoma. The trachomatous material filtered through Berkfeld's candle gave in sixteen experiments a negative result. Addaris placed a trachomatous graft in the inferior cul-de-sac of three blind individuals. Symptoms appeared in a week, and in six or seven weeks trachoma had developed in *both* eyes of all three. Addaris concluded that trachoma was contagious, in spite of the fact that the causal micro-organism has so far not been discovered.

The Diagnosis between Trachoma and Follicular Conjunctivitis.—Many authors deny the distinction, but the discussion showed that most ophthalmologists do differentiate two separate diseases. Follicular conjunctivitis was held to be non-contagious, it leaves no sequelae, and disappears even without treatment. Samperi notes the different localization of the disease: trachoma in the upper lids, follicular conjunctivitis mainly in the lower. Mayweg, v. Graefe, and Samperi failed to induce follicular conjunctivitis by inoculation. Patella attributes the follicular disease to the irritation of dust, wind, smoke, salt water, etc.

Prophylaxis.—Trachoma in Italy and Sicily is frequent among the poorer classes living under bad hygienic conditions. It increases by contagion, owing to the ignorance of the peasants. The notification of trachoma should be compulsory. All candidates for office should be certified as free from trachoma. Out-patient departments should be opened in all the provinces where the disease prevails. There should be separate schools for trachomatous children. All schools should be inspected, and pamphlets explaining the disease should be distributed among the people. Post-graduate courses should be given on trachoma in the universities. Separate wards should be provided for sufferers from trachoma. Trachoma is very prevalent in Malta.

T. HARRISON BUTLER.

In this paper Major ELLIOT (324, **Conjunctival asthenopia due to glare**) of Madras describes the symptoms of glare conjunctivitis which he has frequently noticed in India and which is a well-known disease in Russia, where it is caused

by the glare from snow, and is found in Europe among those who work with the Finsen light and X-rays. The patient complains that "he cannot face glare," when he goes out in the sun he has intense pain and lacrymation. There is much blepharospasm and in a bright light vision is so reduced that the sufferer cannot do his work. Great relief is experienced when he comes into the shade, but frequently some pain persists. There may be chronic headache. On everting the eyelid a slight amount of conjunctivitis is observed; this is frequently *confined to the retrotarsal folds.* This conjunctivitis although so slight is the pivot point of the case. In order to cure the cases the retrotarsal fold must be attacked. Pagenstecher uses copper sulphate stick applied gently. Elliot uses argyrol in from 3%–12% solution. This may be combined with 1% yellow oxide ointment.

The cause is probably purely *intensity* of light, and not quality, or wave length, for it is caused by X-rays which have no wave length. Nor is a preponderance of blue or ultra-violet rays responsible, for the disease is found in light-house keepers whose lights are poor in violet rays.

T. Harrison Butler.

Harrison Butler (325, **The clinical features, bacteriology, and treatment of acute ophthalmia in the East**) describes the epidemic of acute muco-purulent conjunctivitis which every summer descends upon the lands of the Near East, destroying hundreds of eyes. Analyzing 5700 cases seen in Jerusalem he finds that 30% of all cases coming to an oriental eye clinic are sufferers from the disease or its sequelæ.

37,000 new cases yielded 5700 cases of acute ophthalmia, 222 of which had corneal complications. 46% of all ulcers were perforated. Over 200 cases were investigated bacteriologically. Acute ophthalmia was found to have no connection with trachoma, but each was a disease *sui generis.* The chief micro-organisms found were, the diplobacillus of Morax-Axenfeld in chronic and winter cases; the Koch-Weeks bacillus and the pneumococcus in the summer epidemic; and the gonococcus, which was responsible for a large number of corneal ulcers. In the acute epidemic the Koch-Weeks was the cause of 66% of the cases, the pneumococcus of 22%.

Severe ulcers were caused by: the gonococcus 40%; mixed infections 25%; pneumococcus 15%; Koch-Weeks bacillus 10%; Morax-Axenfeld diplobacillus 5%. 5% were apparently sterile. The gonococcus is endemic, and ***exists in Palestine as a purely eye organism.*** Gonorrhœa is practically unknown among the patients who came to the Jerusalem clinic suffering from gonococcal ophthalmia. Ophthalmia neonatorum is never seen. The epidemic always commences at the end of June, and culminates in August. From August to February the case incidence falls rapidly, but the virulence is greatest in November. The ophthalmia curve to a certain extent follows the curve of mean temperature, but it rises four months after the chief rise of temperature. The curves of mean temperature and of ophthalmia percentages fall together.

The disease is certainly carried from eye to eye, by direct contact and by flies.

The symptoms are very acute, and all four organisms can give rise to identical clinical appearances. A severe case which only contains the Morax bacillus may be identical with one caused by the Koch-Weeks or the pneumococcus. On the other hand in the summer epidemic all cases of gonococcal infection are very severe and have corneal complications. Koch-Weeks cases are more likely to be characterized by sub-conjunctival ecchymoses, pneumococcal cases by chemosis. Some of the latter show a pseudo-membrane.

Severe ulcers with hypopyon are seen in Palestine caused by the Koch-Weeks and Morax-Axenfeld organisms. The latter is sometimes intracellular.

Protargol in 33% solution gave the best results, but occasionally silver nitrate in 2% solution was used. The former is best for Koch-Weeks bacilli, and pneumococci in a lesser degree, but the gonococcus often yields to silver nitrate when protargol has failed. Argyrol was found to be comparatively inert. Zinc sulphate in 1% solution is useful in diplobacillary infections.

A bold Saemisch section gives the only hope of saving a severe gonococcal ulcer in Palestine.

The paper contains a large number of charts and statistical tables. T. HARRISON BUTLER.

McKee (326, **A new pathogenic micro-organism of the conjunctival sac**) has isolated what he believes to be a new germ of the conjunctival sac which resembles the influenza bacillus but differs from it widely on media, in viability and pathogenicity. He has only nine clinical cases in which it was present. It grows best upon hemoglobin-agar and blood serum, does not produce gas nor ferment sugar. It is Gram negative, is shorter and thicker than the Koch-Weeks bacillus and thicker than the influenza bacillus. It has no capsule—is non-motile and does not form spores. It is pathogenic for mice, causing septicæmia. ALLING.

Crisp (327, **Chancroidal conjunctivitis**) describes a patient who inoculated his conjunctiva by rubbing the eye with his fingers when suffering from chancroidal ulcers of the penis. The eye developed a violent conjunctivitis with muco-purulent discharge and later was lost through ulceration of the cornea. No gonococci were found in the discharge. ALLING.

Johnston (328, **Pseudo-membranous conjunctivitis caused by staphylococcus aureus and albus**) saw a baby with pseudo-membranous conjunctivitis of one eye following measles. A whitish pseudo-membrane formed on the upper palpebral conjunctiva, which was not closely adherent. Cultures showed no Klebs-Loeffler bacilli, but only staphylococcus aureus and albus.

Both of Krauss's patients (329, **Parinaud's conjunctivitis**) were children and both had been about horses. They showed marked swelling of the retrotarsal folds, which were studded with granulomata, some ulcerated. There was moderate secretion, but great swelling of the lymph glands of the face and neck, especially the preauricular. Bacterial examinations were negative.

Wood (330, **The unilateral form of tarso-palpebral vernal conjunctivitis occurring in a young girl**) excised the nodules and treated the conjunctiva with X-ray with good results. Pusey found the lesions to be confined to one eye in only 7% of cases of vernal catarrh.

Pusey (331, **Involvement of the cornea and bulbar conjunctiva in the secondary stage of syphilis**) finds only twenty-seven cases of secondary syphilitic eruption of the bulbar

conjunctiva and a similar lesion of the cornea is not on record. His case had a general macular eruption with some papules and pustules. The conjunctiva of the lids was also the seat of a pustular eruption. Several of these were located near the centre of the cornea of the right eye. ALLING.

XII.—CORNEA, SCLERA, ANTERIOR CHAMBER.

332. LOEHLEIN, W. **Blood-infiltration of the cornea.** *Deutsche med. Wochenschrift*, 1907, No. 22.

333. V. BARLAY. **Blood-infiltration of the cornea.** *Zeitschrift f. Augenheilk.*, xviii., No. 1, p. 90.

334. TRANTAS. **Superficial keratitis in small-pox and other exanthemata.** *Rec. d'ophth.*, xxix., p. 465

335. LUCARELLI, N. **Sclerosing corneal infiltrate with hyaline.** *Riv. Ital. di ottalm.*, 1907, Fasc. 1–2.

336. LANDMANN. **Two cases of disciform keratitis.** ARCH. OF OPHTH., xxxvi., No. 3.

337. HERTEL, E. **Local phototherapy in ulcus serpens.** *Klin. Monatsbl. f. Augenheilk.*, XLV., No. 2, p. 178.

338. HERTEL, E. **Phototherapy in ocular disease.** *v. Graefe's Arch.*, xlvi., No. 2, p. 275.

339. HESSE, R. **Congestive hyperæmia in ocular therapeutics.** *Centralbl. f. prakt. Augenheilk.*, xxxi., p. 133.

340. DE FALKO, A. **Ocular fatigue in the etiology of pterygium.** *Clin. oculist.*, iv., p. 2805, 1907.

341. SEEFELDER, R. **Peripheral grooving and peripheral ectasia of the cornea.** *Klin. Monatsbl. f. Augenheilk.*, xlv., No. 1, p. 475.

342. ALBRAND, W. **Artificial experimental rupture of sclera.** *Wiener klin. Rundschau*, xxi., 1907, No. 39.

343. VEASEY, C. A. **Primary melanotic spindle-celled sarcoma of the limbus.** *The Ophth. Record*, June, 1907.

344. DUDLEY, W. H. **Carcinoma of the cornea.** *The Ophth. Record*, May, 1907.

345. HUBBELL, A. A. **Cases of congenital inequality in the size of the corneæ in normal eyes.** *The Ophth. Record*, June, 1907.

346. MCKEE, HANFORD. **Ulceration of the cornea from the diplobacillus of Morax-Axenfeld.** *The Ophth. Record*, April, 1907.

347. PATERSON, J. V. **A case of reticular opacity of the cornea (Gittrige keratitis—Haab).** *Ophthalmic Review*, August, 1907.

348. HAY, P. J. **A congenital anomaly of the sclera ("Pseudo-Coloboma").** *Ophthalmoscope*, June, 1907.

LOEHLEIN (332, **Blood-infiltration of the cornea**) describes the microscopic picture of blood-infiltration of the cornea. Descemet's membrane was torn at the periphery and rolled up. Hemorrhage with hemosiderin deposit was found near

Schlemm's canal and in the anterior chamber. Cornea but slightly changed, showed an even uniform slight deposit of hemosiderin, and was crowded with Baumgarten's crystalline bodies.

Barlay (333, **Blood-infiltration of the cornea**) had three cases of corneal hemorrhagic infiltrate after injury. In one, following cataract extraction, blood permeated the cornea from the margins of the section. In the others there was probably imbibition through a pathologically altered Descemet. Iridectomy was without effect.

Trantas (334, **Superficial keratis in small-pox and other exanthemata**) noted the development of keratitis punctata superficialis after measles (76%) and other acute infectious exanthemata.

Lucarelli (335, **Sclerosing corneal infiltrate with hyaline**) considers sclerosing corneal infiltrate an unusual condition. Microscopic examination of excised corneal tissue showed hyaline degeneration with no evidence of amyloid.

Calderaro.

Landmann (336, **Two cases of disciform keratitis**) saw two cases of disciform keratitis with dense central opacity, slightly opaque periphery, and a second more infiltrated margin. There was no previous injury of the cornea, the epithelium was intact, and there was no tendency to necrosis. In one case there was neither pain nor irritation; in the other, intense ciliary irritation was noted with anæsthesis of the opacified area.

Treutler.

Hertel (337, **Local phototherapy in ulcus serpens**) finds that the action of light is not only bactericidal, but stimulates tissues, destroying them after continued application. Ferments and toxines are also inhibited in development. Local hyperæmia and œdema of the eye develop rapidly and increase the bactericidal action of the aqueous. Short-wave-length rays were obtained by magnesium spark, and by cadmium and zinc arc lamp. Ulcus serpens was treated with good results.

Hertel (338, **Phototherapy in ocular disease**) tried various light sources, including induction spark from various metals, carbon arc, sunlight direct and diffuse. Short-wave-length rays affect bacteria only; longer ones also injure tissue.

The reaction is to be carefully distinguished, as in ulcer, from progress of the disease. 27% of ulcus serpens were not improved. The advantage over cauterization consists in the thinner, more transparent scar.

HESSE (339, **Congestive hyperæmia in ocular therapeutics**) used congestive hyperæmia with suction in twenty-three cases of ulcus serpens, excluding all other measures, and noted immediate cessation of pain. In three cases complicated with blennorrhœa of the lachrymal sac, suction hyperæmia was without any effect. There was no marked indication of the ulceration being stopped, but the resulting scar seemed thinner than usual. No data of vision are given.

DE FALKO (340, **Ocular fatigue in the etiology of pterygium**) removes pterygium according to Arlt and Pagenstecher, and in case of relapse implants flaps from a rabbit's cornea. Relapse is due to increased vasomotor reflex movements caused by conjunctival-corneal scar. Atropin relieves the strain and prevents recurrence. The advance of vessels on the cornea is one of the first signs, to be met with strychnin injections in the temple and cauterization of the cornea.

CALDERARO.

SEEFELDER (341, **Peripheral grooving and peripheral ectasia of the cornea**) examined four cases of peripheral grooving and ectasia of the cornea. Microscopic examination showed that the corneal lamellæ as they entered the peripheral region lost their fibrillary structure, becoming more and more homogeneous, more refractive, and finally broke up into irregular homogeneous masses. There is a certain analogy, in a pathologic-histological sense, with arcus senilis, which also occasionally precedes the development of peripheral grooving. The coincident development of pterygium in Seefelder's cases is worthy of notice.

ALBRAND (342, **Artificial experimental rupture of sclera**) experimented on eyes of aged subjects, post-mortem, causing high intraocular pressure by injections through the sclera. In about 12%, scleral rupture took place, generally in the upper inner quadrant, parallel to the limbus corneæ, and about 1–1½*mm* distant from it. Clinically, this intercalary region is also marked by its lack of resistance to tension by disease processes.

VEASEY (343, **Primary melanotic spindle-celled sarcoma of the limbus**) observed a man, forty-seven, who had noticed a pigmented spot at the left limbus for the last twelve years. It suddenly began to grow and in seven weeks covered two thirds of the cornea. The growth was a spindle-celled pigmented sarcoma. About eighty other cases have been reported. They usually occur after middle life, are mostly pigmented, sometimes multiple, and generally are seen in the exposed portion of the limbus between the lids.

DUDLEY (344, **Carcinoma of the cornea**) relates an interesting example of metaplasia of carcinoma of the cornea. Fifteen years after an injury to the eye, a man, eighty-three, presented a vascularized macula near the limbus. One year later the area looked to be papillomatous and sections proved it to be of this nature. At three different times sections were made and all showed this character. Upon the fourth examination the type of cells had changed and was now unquestionably carcinomatous.

HUBBELL (345, **Cases of congenital inequality in the size of the corneæ in normal eyes**) saw two instances of congenital inequality of corneæ in eyes which were otherwise normal, except for a slight degree of hypermetropia. There was a difference in the two corneæ of *3mm* in one case and *1½mm* in the other.

In one hundred cases of conjunctivitis produced by the Morax-Axenfeld diplobacillus MCKEE (346, **Ulceration of the cornea from the diplobacillus of Morax-Axenfeld**) found seven complicated with corneal ulcers. All of the cases yielded to zinc therapy. The writer thinks ½% sulphate of zinc unnecessarily strong in the treatment of these affections. BLAKE.

PATERSON, J. V. (347, **Case of reticular opacity of the cornea**) records a case of Haab's *gittrige keratitis* and his article is illustrated by some very clear drawings of the two corneæ. The patient, a widow, aged forty-eight, was first seen in April, 1907. Sight has failed in the left eye for 2½ years. There was no family history of a similar disease. The left eye was free from injection, the cornea showed a number of anastomosing gray lines which formed an irregular meshwork. In the drawing they rather suggest a photograph of a branched flash of lightning. Towards the

W. Krauss fec.

Krauss-Brückner, Zur Kenntniss der Tuberkulose des Augenhintergrundes.

Kgl. Univers. Druckerei v. H. Stürtz, Würzburg. Verlag v. J. F. Bergmann, Wiesbaden.

W. Freytag gez.

Krauss-Brückner, Zur Kenntniss der Tuberkulose des Augenhintergrundes.

Kgl. Univers.-Druckerei v. H. Stürtz, Würzburg. Verlag v. J. F. Bergmann, Wiesbaden.

centre the lines tend to fuse forming a considerable opacity. There is a faint corneal haze in addition at the centre. The lines lie superficially just under the epithelium, which is raised over them and shows slight stippling. The general haze consists of fine dots, lying deeper in the cornea than the lines. L:V=$\frac{6}{24}$; fundus normal. The right cornea only shows some faint wavy lines. R: V corrected=$\frac{6}{9}$.

Haab (*Zeitschrift für Augenheilkunde*, 1899, p. 236) has figured a very similar case. He looks upon the disease as degenerative, and Paterson agrees with this. Haab's case was a family affection, several members being similarly affected.

Dimmer cites three cases (*Zeitschrift für Augenheilkunde*, 1899, p. 354) occurring in three members of a family, but the drawing given does not correspond with what Paterson has seen in typical cases. BUTLER.

HAY (348, **A congenital "pseudo-coloboma" of the sclera.** This unique anomaly was found in a still-born child, born at full time. The infant had enormous hydrocephalus, double hare-lip, and complete cleft palate. The right ear was malformed, with an imperfect external meatus; there was an accessory auricle on the left side. On examining the left eye there appeared a condition which looked very much like a defect in the sclera. On both sides of the cornea and on a level with the horizontal meridian there was an area as dark as the pupil, triangular in shape, with the base adjoining the cornea. The whole had a lozenge shape. The left eye presented a similar condition, but only on the temporal side. A section of the right eye showed that the sclera was very thin in this lozenge-shaped area; the whole anterior part of the sclera was thin, the posterior very thick, the relations being one to four. Hay regards the condition as being *sui generis* and not buphthalmos. The paper is well illustrated and to fully grasp the condition these figures must be studied.

T. HARRISON BUTLER.